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THE CANADIAN ANAESTHETISTS' SOCIETY JOURNAL

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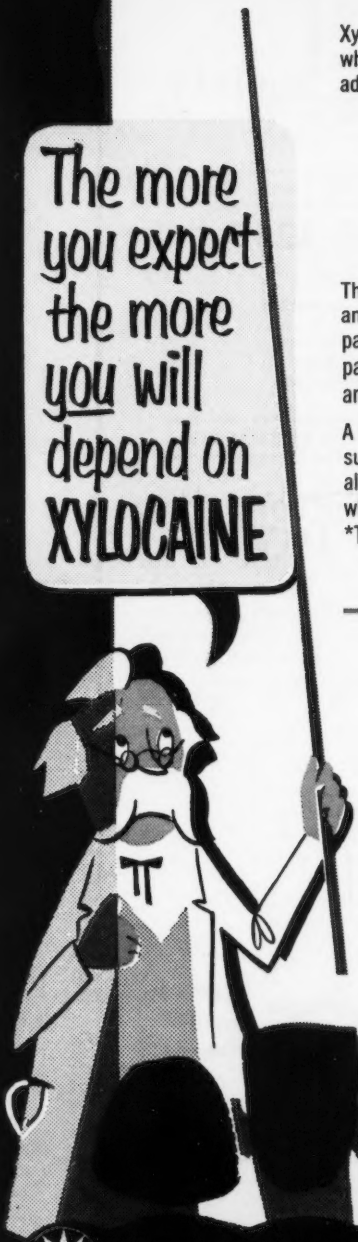

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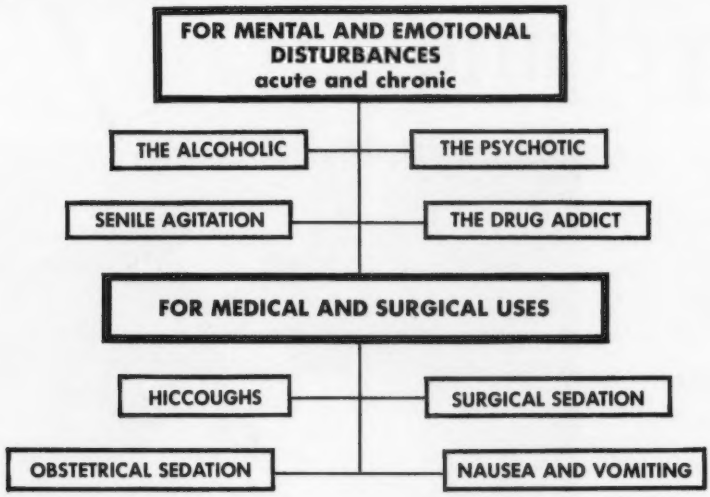

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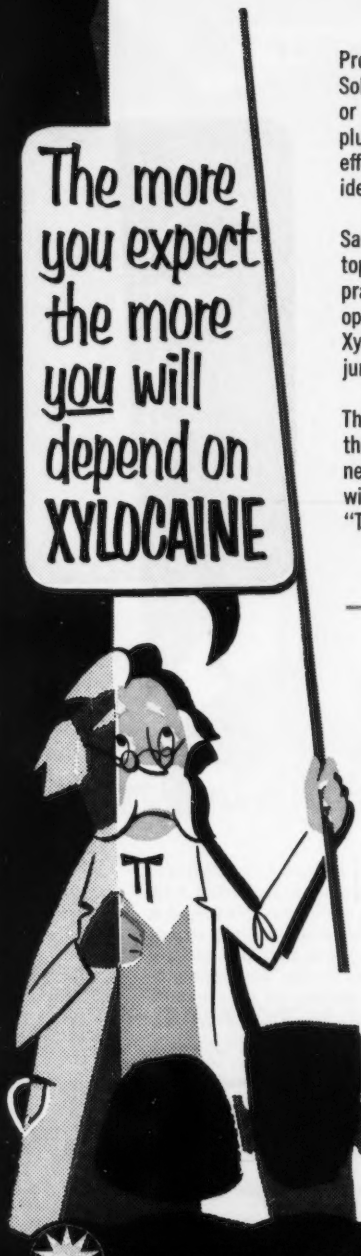

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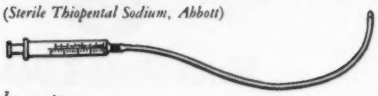

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EDITORIAL

On Mixtures of Anaesthetic Agents

There has been a recurring concept in anaesthesia, inherited undoubtedly from the polypharmacy of the past, that the advantages of agents might be enhanced and their undesirable properties suppressed or minimized by mixture with other agents. This is the basis of the currently popular cult of "balanced anaesthesia" just as much as it was the basis for earlier mixtures of chloroform-ether, alcohol-ether, vinethene-ether, or plain "Mist. Expect. Sed."

While this concept is undoubtedly sound, the practical application of it in the clinical use of mixtures of liquid anaesthetic agents has proved unsatisfactory in the past, either because the mixtures used were physically unstable so that the proportions of the component agents being volatalized at any one time were uncertain, or because the mixtures did not, in fact, materially alter the undesirable characteristics of their components.

Elsewhere in this Journal Dr. Fernando Hudon and his colleagues at Laval University describe the advantages to be gained by the use of an azeotropic mixture of two liquid anaesthetic agents—in this case Fluothane and ethyl ether. These authors also point out that this principle appears to have been previously unexplored in anaesthesia. That this can be true in an age in which gasoline is an everyday commodity must leave us all astounded and not a little abashed Undoubtedly the possible benefits of other similar combinations reuire careful study.

Concernant les mélanges d'agents anesthésiques

En anesthésie, il y a un concept qui revient à la mode, concept qui est sans doute un héritage de l'ancienne polypharmacie et qui veut que les propriétés avantageuses des agents soient améliorées et que les ennuis qu'occasionnent ces agents soient supprimés ou minimisés quand on en fait un mélange avec d'autres agents. Cela est la base du culte populaire actuel de l'"anesthésie balancée" tout comme cela était la base pour les anciens mélanges chloroforme-éther, alcool-éther, vinethene-éther, ou la simple "Mist. Expect. Sed."

Bien que ce concept, sans aucun doute, semble rationnel, son application pratique dans l'emploi clinique de mélanges d'agents anesthésiques liquides s'est avérée non satisfaisante dans le passé, soit parce que les mélanges employés étaient physiquement instables de sorte que, à un moment donné, on ne pouvait pas être certain des quantités volatilisées de chacun des agents en cause, soit parce que, de fait, les mélanges, matériellement, ne modifiaient pas les effets indésirables des agents en question.

Ailleurs dans ce journal, le Docteur Fernando Hudon et ses collègues, de l'Université Laval, font part des avantages obtenus par l'emploi d'un mélange

azeotropique de deux agents anesthésiques liquides: le Fluothane et l'éther éthylique. De plus, ces auteurs affirment que l'application de ce principe en anesthésie n'a pas été l'objet de recherches antérieures. Que cela soit conforme à la vérité dans un temps où la gazoline est devenue une commodité quotidienne, il y a lieu d'être surpris et pas le moindrement confus. Sans aucun doute, il s'impose de faire une étude attentive de d'autres mélanges semblables pour en découvrir les avantages possibles.

SERIAL CARDIAC OUTPUT DETERMINATIONS IN MAN[1]

J. E. Merriman, F.R.C.P.(C), G. M. Wyant, F.F.A.R.C.S.,
G. Bray, M.D., and W. McGeachy, M.D.

The ability to measure cardiac output accurately under various conditions determined by anaesthetic agents and techniques and by surgical stress is of obvious interest. Even more important is the availability of a simple and reliable method of measuring serial cardiac outputs and thus of monitoring changes in cardiac dynamics. This information, when correlated to blood pressure readings, electrocardiograms, etc., will afford a much clearer picture of the reaction of the cardiovascular system to changing stress, than blood pressure, pulse, or electrocardiographic studies alone.

In order to be of clinical use, a method of serial cardiac output determination must be reasonably accurate, technically not too complicated, and not be overly time-consuming. It must require a minimum of expensive apparatus and technical personnel. Furthermore, such a method must not be harmful to the patient, must not increase the stress of surgery and anaesthesia significantly, and must leave no undesirable after-effects. To understand how these criteria may be fulfilled, it is best to consider briefly the various methods of cardiac output determination which have been developed in the past and to examine how they can be best adapted to serial determinations within these criteria.

There are three main groups of tests available, the physical methods, the gasometric methods, and the indicator-dilution methods.

Physical Methods

Analysis of the *pulse wave contour* has shown that the area under the aortic pressure curve is proportional to the stroke output and a factor related to the volume-elasticity characteristics of the vessel. It is possible to integrate these pressure curves electronically. If one were interested only in relative changes and not in absolute values, this method might have some merit for serial determinations. We have made only preliminary observations with this method.

The *ballistocardiograph* is actually a measure of cardiac force, but, because of the relationship between cardiac force and cardiac output, it has been used as a method for determination of cardiac output. Similarly, the *pneumotachygram* and the *impedance cardiogram* have not been found to be satisfactory by investigators in this field and none is in use at the present time as far as we are aware.

Gasometric Methods

Direct Fick

This method is generally accepted as the standard method for determination of cardiac output with which all newer methods must be compared. Determinations are made from the following formula:

$$\text{Cardiac output (L./min.)} = \frac{\text{O}_2 \text{ consumption (ml./min.)}}{\text{Pulmonary A-V O}_2 \text{ difference (ml./L.)}}.$$

[1]From the Departments of Medicine and Anaesthesia, College of Medicine, University of Saskatchewan, and Cardio-Pulmonary Laboratory, University Hospital, Saskatoon, Saskatchewan.

The difficulty of obtaining samples of mixed venous blood limits the use of this method. The samples are obtained by means of cardiac catheterization with the catheter tip placed in the main pulmonary artery. It is at once obvious that a technique for cardiac output determination which requires cardiac catheterization cannot easily be adopted as a routine procedure. The cardiac catheter must be introduced under fluoroscopic and pressure control. Positioning of the catheter tip in other than the outflow tract of the right ventricle or in the main pulmonary artery is undesirable since the mixed venous blood is the major variable in the Fick determination. This is so because of the inadequate mixing of coronary sinus blood in positions other than the ones mentioned.

For the greatest accuracy, all three samples in the equation must be collected over the same period of time, and therefore the final answer is a mean determination of cardiac output over the period of collection. This is usually the three minutes required for the collection of expired air.

It is apparent that the Direct Fick method, while accurate, is time-consuming. It does not lend itself readily to serial determinations under anaesthesia in man, because maintenance of the catheter tip in the same position is uncertain and because repeated air-sampling will interfere with maintenance of inhalation anaesthesia.

Indirect Fick Procedures

Krogh and Lindhard (1) first pointed out that measurements of cardiac output could be made from the rate at which a foreign gas of known concentration in the lungs is taken up by the blood. Several of the earlier reports of cardiac output determination were done by this method, using as the test gas either nitrous oxide or acetylene. This method has several obvious disadvantages for the determination of serial cardiac output, especially during anaesthesia.

Osborne *et al.* (2) have recently described a method which utilizes the lung as a tonometer. When rapid carbon dioxide analysers and different concentrations of carbon dioxide are used, it is possible to determine the mixed venous and arterial carbon dioxide tensions, as well as the total carbon dioxide production. This method does not require the co-operation of the patient and can be made over a period of 8 to 10 seconds.

Indicator-Dilution Methods

The principle involved in all these methods is the same. An indicator in injected rapidly into the circulation and its subsequent concentration in the arterial blood is checked at frequent time intervals.

The indicator may be either a source of radioactivity or a foreign material, such as a dye. A scintillation counter is required to record the concentration of radioactive materials. The concentration of dyes in the arterial blood can be measured either by the collection of serial arterial samples, or by the withdrawal of arterial blood through a cuvette oximeter. The resulting dilution curves are then analysed and the cardiac output determined.

RADIOACTIVE ISOTOPES

Veall and associates in 1954 (3) and Huff and co-workers in 1955 (4) published methods of determining cardiac output in man by the *in vivo* analysis of radioactive iodinated human serum albumin (RIHSA). They were able to carry out a time-intensity analysis of I^{131} with a highly shielded well-collimated scintillation detector placed over the chest. They advocated the use of 100–200 microcuries and went as high as 300 microcuries for repeated tests. The curves thus obtained, when analysed for cardiac output, gave values not significantly different from Direct Fick values obtained simultaneously. In seven patients and twenty-two dogs tested by the radioactive method and simultaneous Fick determinations, the means were not significantly different.

The technique for these tests is as follows. The detector is placed over the skin between the first and second ribs, immediately to the left of the sternum. This position is selected since here the outflow tract is closest to the anterior chest wall with a minimum of interposed tissues, since the blood flow through tissue would alter the radioactivity transient. The scintillation counter is connected to a count-rate meter and a strip-chart recorder. We have found doses of 75 microcuries quite adequate to obtain good curves and these need not be exceeded. As with all indicator-dilution methods, the indicator must be injected rapidly and immediately flushed with normal saline. The calculation of cardiac output from this curve is done by standard methods. Since the radioactive material is carried in plasma, knowledge of the plasma and blood volumes is essential. The total blood volume has been previously determined using I^{131} for plasma and Cr^{51} for red cell volumes.

The major disadvantage of this method for serial determinations is the relatively large dose of radioactive material needed per injection. Although potassium iodide is administered before and after the tests to block the thyroid, we believe that a total dosage of 300 microcuries should not be exceeded. Thus one is limited to approximately four determinations. A reduction in the amount of radioactive material injected would lead to a markedly flattened curve with greater errors in the results. This problem could be resolved by using a more sensitive detector of radioactivity.

The use of this method in the operating room during anaesthesia presents still further disadvantages. First, the detector which we have been using is bulky. Secondly, for accurate determination of cardiac output it is necessary to know the exact blood volume of the patient at the time of each determination. This is simple in the experimental laboratory, but in the operating room, where blood loss and blood replacement are constantly proceeding, repeated determinations of blood volume will be necessary. This is relatively easy once plasma and red cells have been tagged initially, provided blood loss and replacement are known approximately. However, it would be advantageous to have a method of cardiac output determination which is independent of changes in blood volume.

In summary, then, this is a satisfactory method, but further technical advances, especially in the construction of scintillation counters, are needed before it is an ideal method for serial cardiac output determinations, especially during operations.

Dye Dilution Methods

Evans Blue in Plasma

A modified method for determining cardiac output by this means was described by Etsten and Li in 1954 (5). In brief, the method consists of the injection of a precise amount of T-1824 (Evans Blue) in ½ per cent solution into the median basilic vein followed by a 10 ml. saline flush. Samples of arterial blood are collected, at the conclusion of the intravenous injection of dye, in 32 small glass tubes attached to a circular plastic disc. The disc is rotated by a motor in clockwise fashion at a speed of one tube per second. The arterial blood samples are centrifuged and the plasma transferred to individual microcuvettes. The dye concentration of each microcuvette is read on a Coleman spectrophotometer at a wave length of 625 mμ against the plasma control in the first tube. The optical densities are translated into dye concentrations in milligrams per litre from a previously prepared plasma dye curve. These dye concentrations are plotted against time in seconds on semi-logarithmic paper. The cardiac output is determinted from the dye concentration curve according to the method of Hamilton (6) and Eliasch (7):

$$F = \frac{60 \times I}{c\,t} \times \frac{100}{100 - H}$$

where F = cardiac output in litres per minute
I = amount of injected dye in milligrams
c = mean concentration of dye in milligrams per litre
t = passage time of dye in seconds
H = haematocrit (not corrected for trapped plasma)

Etsten has shown that the values obtained by this method closely approximate those obtained with the Direct Fick method, if special attention is paid to a number of minor points mainly connected with the technique of injection of the dye. This method depends upon a free flow of arterial blood so that each tube contains an equal amount of blood. We have determined the time needed for each determination including centrifuging and pipetting off of plasma. In our laboratory it takes two technicians a total of 2½ hours to make one output determination. This time factor, then, is a real handicap for rapid serial determinations.

Evans Blue is slowly excreted from the body, and although it begins to leave the plasma within ten minutes of injection, it is held for a long time in the reticuloendothelial system. Clinically this means that the patient will look increasingly blue and may continue to do so for several days or even weeks with dye being slowly excreted; this may give rise to blue-stained urine. The total blood loss incurred from repeated determinations is not insignificant, since approximately 40 ml. of blood are withdrawn for each test. Lipaemia and haemolysis both interfere with dye determinations in the Etsten method.

For all these reasons the Etsten method for serial determination of cardiac output in man is impractical.

Evans Blue in Whole Blood

In order to obviate at least some of the disadvantages of the Etsten method, and to reduce the call on technicians' time, the method was modified in this

laboratory by Rainbow and McGeachy. Since this has not been described in the literature, it will not be discussed here in detail. Suffice it to say that two technicians working together can complete the analysis of forty tubes in one hour. In normal subjects this method has been found satisfactory and, with normal arterial oxygen saturation, the effect of haemoglobin at 625 mμ is minimal. Simultaneous determinations of cardiac output in volunteers with this and the original Etsten method show that the results are closely similar. They have been checked against the methods using radioactive isotopes and again results were comparable.

This modification shares with the Etsten method the disadvantage that the subjects become increasingly blue. The method also has inherent difficulties arising from maintenance of free arterial blood flow, and blood loss is by no means insignificant if a large number of determinations are done.

Indicator Dilution Curves Using Cardio-Green

The use of Cardio-Green obviates two of the great disadvantages of Evans Blue. (i) Since Cardio-Green does not accumulate in the body, repeated injections of the dye do not lead to discoloration of the patient. (ii) A monitoring cuvette system can be employed with a consequent saving in the time needed to analyse a series of individual samples and construct a dye-dilution curve from the results. Such a cuvette system gives unreliable results with Evans Blue since the system is sensitive to both dye concentration and changes in the percentage of oxygen saturation of the blood. Thus the curves are uninterpretable in the presence of fluctuations in oxygen saturation. Cardio-Green does not have this disadvantage.

Fox and his co-workers have reported their studies with a tricarbocyanine dye which later has been given the trade name of Cardio Green (8). This dye has a peak concentration at a wave length of 800 mμ, a point at which the spectral transmission curves of oxyhaemoglobin and reduced haemoglobin intersect. This means that with this dye variations in oxygen saturation have no effect.

With the cuvette oximeter recording system in the determination of indicator-dilution curves using Cardio-Green, only the infrared cell of the cuvette is used. This is sensitive at a wave length of 800 mμ.

Preliminary information reveals that Cardio-Green travels with the plasma proteins. Personal communication with several investigators has failed to reveal any instance of toxicity even in doses as high as 2 mg./kg.

In order to confirm that the dye is non-toxic, we have carried out a number of laboratory tests on eight volunteers on whom not less than six and as many as ten consecutive cardiac output determinations have been done. Tests included haemoglobin estimation, packed cell volume, and total and differential white blood counts. Liver function tests done were thymol flocculation, thymol turbidity, zinc sulphate flocculation, and blood albumin-globulin determinations, including electrophoresis for the various globulin fractions. A urinalysis including reaction, specific gravity, sugar, acetone, protein, and microscopic examination was also carried out. These tests were done both before and 48 hours after the cardiac output determinations. None of the tests showed any significant changes except in one subject who had had a pre-anaesthetic elevation of gamma globulin,

thymol turbidity, and zinc sulphate turbidity; he showed a further rise of zinc sulphate turbidity on the day following the cardiac output determination. At the same time he also had a double plus thymol flocculation test. All these were repeated six days later and had by then returned to pre-experimental levels.

For the above reasons this method, using Cardio-Green as the indicator and the modified cuvette oximeter system as the recorder, was found to be the most suitable for our purposes for cardiac output determination.

Method. The volunteers were given a complete physical examination and those with significant diseases were excluded. Eight healthy subjects were chosen. They came to the laboratory in a fasting state. Cardio-Green was used in a concentration of 5 mg./ml. Figure 1 shows our injection apparatus.

The dye dilution curve was obtained as follows. Using an oiled 30 ml. syringe, blood was withdrawn at a steady rate from the artery through the cuvette oxi-

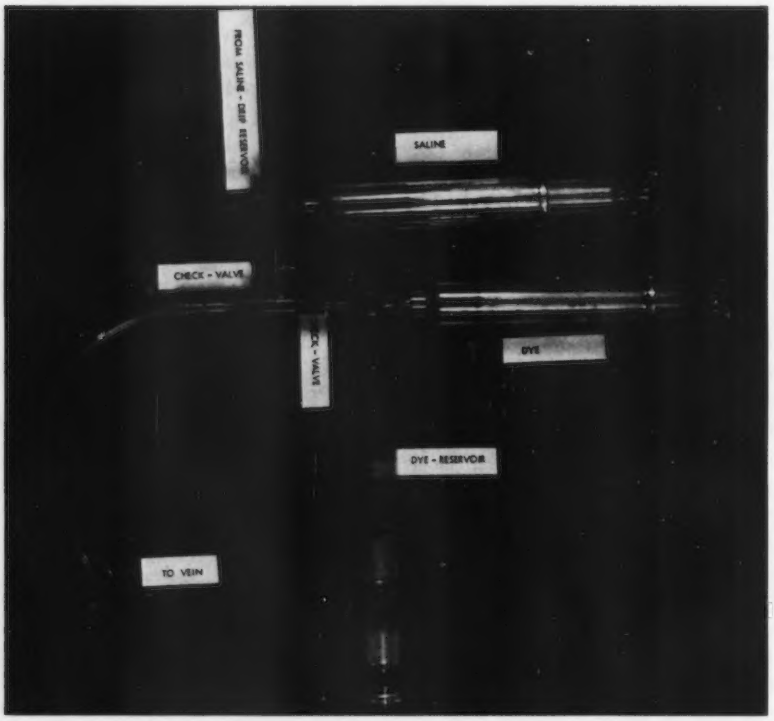

FIGURE 1. Injection apparatus for Cardio-Green. The apparatus consists of two one-way check valves. One limb is connected by a three-way stop cock to two syringes containing Cardio-Green. One of these is used as a reservoir syringe; the other contains the measured dose of dye. The other limb of the apparatus is connected by a three-way stopcock to a syringe containing saline and to an intravenous drip which acts as a saline reservoir. The whole unit is connected by a short segment of polyethylene tubing to the needle in the antecubital vein.

meter. The light beam of the oximeter recorder was then put at zero and the signal for injection given. The measured dose of dye was injected quickly with one hand and a flushing dose of 10 ml. of saline was rapidly injected with the other hand. Injection and flush were signaled on the oximeter recorder. The withdrawal of arterial blood through the cuvette was continued at a constant rate and each millilitre of withdrawal was recorded on the oximeter record.

Meanwhile, the light beam of the oximeter recorder was followed visually while being recorded on instantly developing photographic paper. When the plateau following the re-circulation hump was reached, the withdrawn blood was re-injected. Thus the net loss of blood to the patient was negligible. The equipment was then flushed with saline. The actual curve could be inspected within one minute of its completion. With practice an estimate of the cardiac output could be made immediately, but the actual curves were measured accurately later.

The Hamilton method (6) for calculation of cardiac output was used. Since the disappearance or clearing of the injected dye occurs exponentially, the descending limb of the curve was extrapolated on semi-logarithmic paper until a theoretical point of zero concentration was reached. This in actual practice was taken as 0.1 mg./L. The area under this extrapolated curve was then determined, using a Keuffel and Esser planimeter, Model 4242. The area in square centimeters was then divided by the length of the base, giving a height which, when measured on the calibration curve, gave the mean concentration of dye in milligrams per litre. Cardiac output was then determined by the previously mentioned formula:

$$F = \frac{60 \times I}{c\,t}.$$

That part of the calculation which refers to haematocrit is omitted, since Cardio-Green determinations are not influenced by changes in haematocrit.

Figure 2 shows some typical cardiac output curves in the same subject with both normal and reduced cardiac output, and using different amounts of dye.

This method of determining cardiac output has served us well, both in experimental work and in determinations on patients with various kinds of heart disease.

Summary and Conclusions

Of all the methods available for the serial determination of cardiac output, the one using Cardio-Green has proved the most suitable. Most other methods of cardiac output estimation do not lend themselves readily to many consecutive tests, with the exception perhaps of the ones using radioactive tracers. The suitability of the latter, however, depends upon the availability of highly sensitive counters so that the total dose of radioactive material injected during all these tests does not reach dangerous levels.

Résumé

Nous avons étudié les diverses méthodes à notre disposition actuellement pour mesurer le débit cardiaque et nous avons discuté de l'utilité de chacune pour pratiquer des déterminations en série.

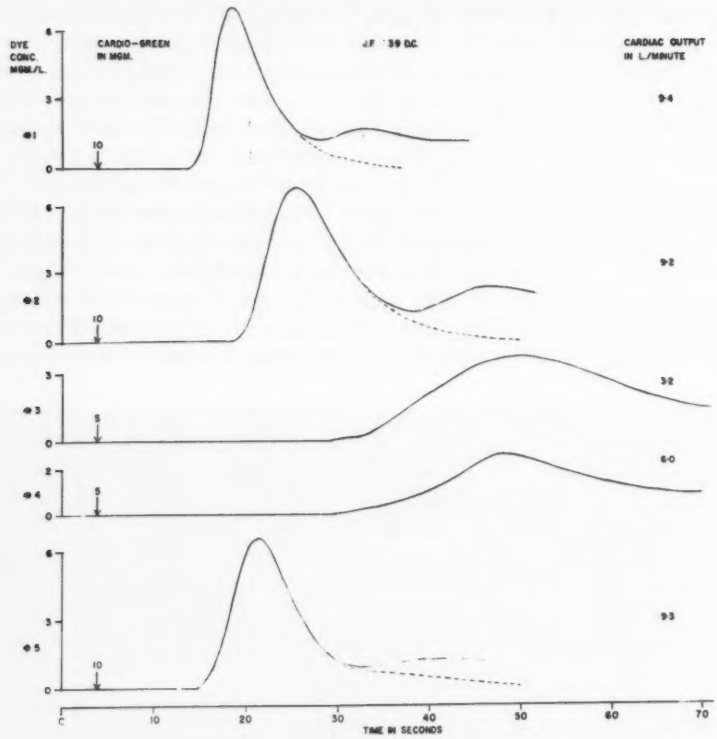

FIGURE 2. Typical curves of different cardiac outputs with different doses of dye.

On peut diviser en trois groupes les méthodes de mesurer le débit cardiaque: des méthodes physiques, gazométriques, et de dilution d'indicateur. Les diverses méthodes physiques comprennent l'analyse du tracé du pouls, la ballistocardiographie, la pneumotachygraphie, et l'impédance cardiographique. Les méthodes gazométriques comprennent la détermination directe de Fick et un certain nombre de procédés indirects de Fick. En ce qui concerne les méthodes de dilution d'indicateur, on emploie soit des substances radioactives, soit des teintures. On a fait usage à maintes reprises du Bleu Evans pour déterminer des débits cardiaques, mais son emploi n'est pas pratique pour faire de nombreux tests consécutifs. Le Vert-Cardio, une teinture tricarbocyanine, possède plusieurs avantages sur le Bleu Evans dont les plus importants sont de ne pas s'accumuler dans l'organisme et de ne pas être toxique. On peut donc, en conséquence, chez le même individu, l'employer à répétition à de courts intervalles. On obtient les courbes de dilution rapidement à l'aide d'un système moniteur oxymètre cuvette, car la courbe définitive ne subit pas l'influence des variations de la saturation en oxygène du sang.

Nous avons décrit, en détail, la façon exacte d'employer le Vert-Cardio car elle est devenue notre méthode de choix pour déterminer le débit cardiaque

chez l'humain. Le seule autre méthode convenable est celle de l'usage de matériel radioactif, à la condition d'avoir des compteurs très sensibles pour dépister la radioactivité de façon à ce que la radioactivité totale n'atteigne pas des proportions dangereuses.

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THE CARDIOVASCULAR EFFECTS OF HALOTHANE[1, 2]

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ONE OF THE INTERESTING and disturbing properties of halothane—$CF_3CHClBr$ (Fluothane®)—is the ease and rapidity with which it can produce hypotension which is sometimes of an alarming degree. It has been variously speculated that this might be due to either peripheral vasodilatation or myocardial depression, or to a combination of both. Hitherto, experimental evidence for either viewpoint has been lacking. This study was undertaken to elucidate the effects of halothane upon the cardiovascular system.

METHOD

The experiments were carried out on healthy male volunteers. The men were instructed not to breakfast on the day of the experiment. After admission to the Recovery Room, blood and urine specimens were taken. Haemoglobin, packed cell volume, total and differential white counts, and a routine urinalysis, including reaction, specific gravity, sugar, acetone, protein, and microscopic examination, were done. Liver function was evaluated by the thymol flocculation, thymol turbidity and zinc sulphate flocculation tests and by blood albumen-globulin determinations, including electrophoresis for the various globulin fractions. These tests were repeated the day following the experiment.

No premedication was administered. A cardiac catheter was passed, under fluoroscopic control, from the left median cubital vein into the pulmonary artery. An intravenous needle was placed into the median cubital vein of the other arm, and a 20-gauge Riley needle was inserted into either the brachial or the femoral artery. Electrocardiographic and electroencephalographic leads were applied.

Cardiac output. Cardiac output was determined by the indicator-dilution technique, using the venous injection of Cardio-Green[4] with arterial sampling through a cuvette oximeter (1, 2). The exact method has been described in a separate publication, which should be consulted for details (3). The advantages of using Cardio-Green and oximetry, as opposed to other dye methods, are that (*a*) there is no significant blood loss, (*b*) results are almost immediately available, (*c*) the method requires a minimum of the technicians' time for the calculation of output, (*d*) any number of determinations can be carried out without accummulation of dye, and without toxic effects, and (*e*) variations of percentage of oxygen saturation have no effect on the output curves.

Peripheral arterial pressure. By means of the indwelling arterial needle, con-

[1]Presented at the Annual Meeting of the Canadian Anaesthetists' Society, Seigniory Club Montebello, P.Q., June 23–25, 1958.

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tinuous arterial pressure tracings were recorded in a manner similar to that described in a previous publication (4).

Pulmonary artery pressure. The same recording devices were used for pulmonary artery pressure as for peripheral arterial pressure. The cardiac catheter was connected to a Statham strain gauge and thence to a recording device which allowed continuous monitoring and recording of these pressures.

Electrocardiogram. A third channel on the recording device was used for continuous monitoring and recording of the electrocardiogram.

Electroencephalogram. Continuous tracings were taken during the experiment by means of an Edin Anesthograph.

Vein-to-artery circulation time was the "appearance time" of the cardiac output curve: that is, the time which elapsed from injection of the dye into the vein to its appearance in the arterial blood, as this was drawn through the oximeter cuvette.

Total peripheral resistance was calculated from the following formula:

$$R = \frac{B(F)A_m - O}{CO} \times 1{,}332$$

where R = total peripheral resistance (Dynes/sec./cm.$^{-5}$)
$B(F)A_m$ = brachial (or femoral) arterial mean pressure (mm.Hg)
CO = cardiac output (ml./sec.)
O = approximation of left ventricular end-diastolic pressure
1,332 = 980 (gravity factor) $\times$ 1.36 (specific gravity of Hg) for conversion to absolute units.

Total pulmonary resistance was calculated from a similar formula, substituting PA_m (pulmonary mean arterial pressure in mm. Hg) for $B(F)A_m$.

Control values were recorded over a period of not less than ten minutes, and at least two control cardiac ouput measurements, closely similar to one another, were obtained. Anaesthesia was then induced with small incremental doses of 2.5 per cent thiamylal sodium. This was done in order to avoid the subjective unpleasantness of a slow inhalation induction, and to prevent the excitement stage during which the many needles might have been dislodged. The amounts of thiamylal used were never sufficient to produce either apnoea or hypotension. Halothane was then administered from a Fluotec® vaporizer in a semi-closed (partial rebreathing) system using a flow of 10 L./min. of oxygen. The concentration of halothane was gradually increased during the first ten minutes or so to the maximum of 3 per cent. This concentration was maintained until all vital signs had become stabilized. An oropharyngeal airway was inserted as soon as it could be tolerated. Endotracheal tubes were not used. When the blood pressure had been stable for at least five minutes, halothane was administered in a closed system with carbon dioxide absorption, a reduced flow of oxygen and controlled respiration—the vaporizer being left full on—until the maximum hypotension compatible with safety had been attained. In one or two patients blood pressure was allowed to fall as low as 30 mm. Hg

[4]Kindly supplied through the courtesy of Dr. John H. Brewer, Director of Biological Research, Hynson, Westcott & Dunning, Inc., Baltimore, Maryland, U.S.A.

systolic. Cardiac output determinations were done at various stages of anaesthesia. After cardiac output determinations had been completed at the maximum hypotension, halothane was discontinued, oxygen administered in a semi-closed system at 10 L./min., and the subject allowed to waken spontaneously. Cardiac output determinations were done again during various stages of awakening, the last one just before the subject answered to verbal command, but when he was still lying quietly.

At various stages of the experiment, the effects of a number of agents on blood pressure, cardiac output, etc., were observed. Atropine was given to two subjects after moderate hypotension had been reached. In four subjects phenylephrine was administered intravenously when the blood pressure had reached its lowest level. One volunteer, who had had neither atropine nor phenylephrine, was given Lanatoside C when a moderate degree of hypotension with a nodal rhythm existed during awakening.

Results

Subject I (Fig. 1)

Induction was smooth, but the systemic arterial pressure rose from 125/75 mm. Hg to 160/85 mm. Hg and the pulmonary artery pressure from 21/8 mm. Hg to 26/16 mm. Hg. A concentration of 3 per cent halothane was attained within 12 minutes.

Anaesthesia was maintained at this level for 20 minutes during which time some peripheral vasodilatation was noted, and the blood pressure fell rapidly to 80–90 mm. Hg systolic and then levelled off. Similarly the pulmonary artery pressure fell to 17/9 mm. Hg. and stabilized at its induction level. The cardiac output by this time had fallen from a control value of 9 L./min. to 7 L./min. and the vein-to-artery circulation time had increased from 11 to 13 seconds. The total peripheral resistance had declined somewhat, but the total pulmonary resistance was markedly raised.

When these observations had been completed, the administration of halothane was continued in a closed system with carbon dioxide absorption. It quickly became necessary to assist and then control ventilation. There was now a slowly progressive further fall of pressure. But neither the cardiac output nor the vein-to-artery circulation time had changed significantly at a time when the systemic blood pressure had reached 60/40 mm. Hg. The total peripheral resistance declined, and total pulmonary resistance fell again.

When the systemic blood pressure had fallen to 45/30 mm. Hg, the cardiac output had also fallen to 5 L./min. Meanwhile, the pulmonary artery pressure had steadily declined to 14/6 mm. Hg, vein-to-artery circulation time had increased to 14 seconds, the total peripheral resistance had risen slightly and total pulmonary resistance markedly.

The systemic blood pressure rose sharply to 130/80 mm. Hg as soon as halothane was discontinued.

No abnormalities were seen in the electrocardiogram during the experiment.

Subject II (Fig. 2)

During the induction, which was smooth, there was a temporary fall of systemic blood pressure from 140/70 mm. Hg to 120/70 mm. Hg, accompanied by a fall of pulmonary artery pressure from 25/15 mm. Hg to the control level (18/9 mm. Hg). A concentration of 3 per cent halothane was reached within 8 minutes.

The blood pressure dropped sharply 8 minutes later to 100 mm. Hg systolic and then rose to a steady level of 110/50 mm. Hg. At the same time the pulmonary artery pressure rose to 28/15 mm. Hg and then stabilized at 24/15 mm. Hg. At this time the cardiac output had fallen to 7.7 L./min. from pre-anaesthetic values of 11

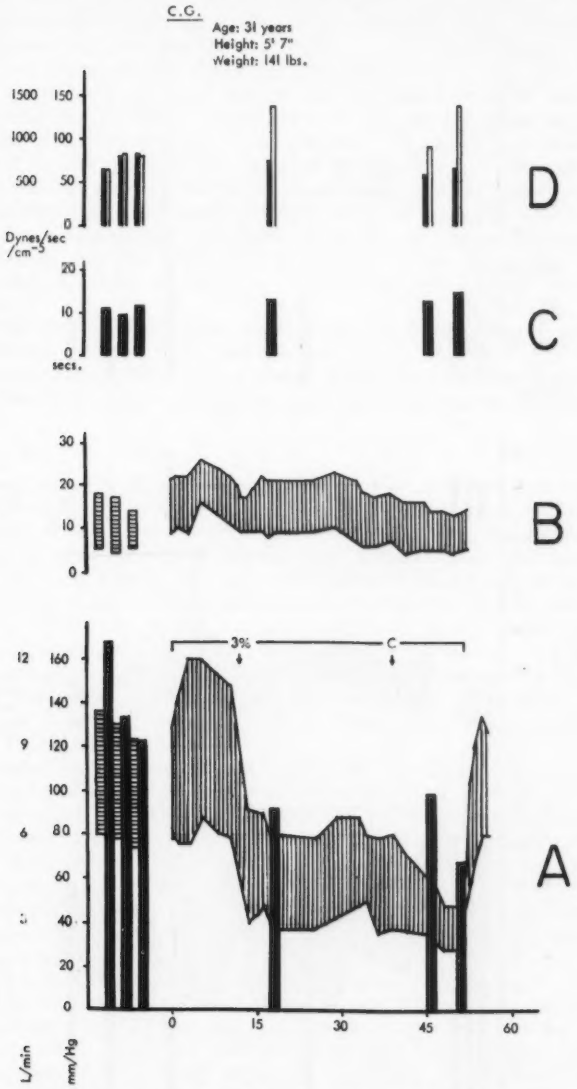

FIGURE 1. *A*, systemic blood pressure in mm.Hg (pulse pressure shaded). Cardiac output (black bars) in L./min. Bracket denotes duration of administration of halothane; the time when 3% concentration was reached and when the system was closed is marked by "3%" and "C" respectively. *B*, pulmonary artery pressure in mm.Hg. *C*, vein-to-artery circulation time in seconds. *D*, total systemic resistance (black bars; scale on left); total pulmonary resistance (white bars; scale on right); both—Dynes/sec./cm.$^{-5}$

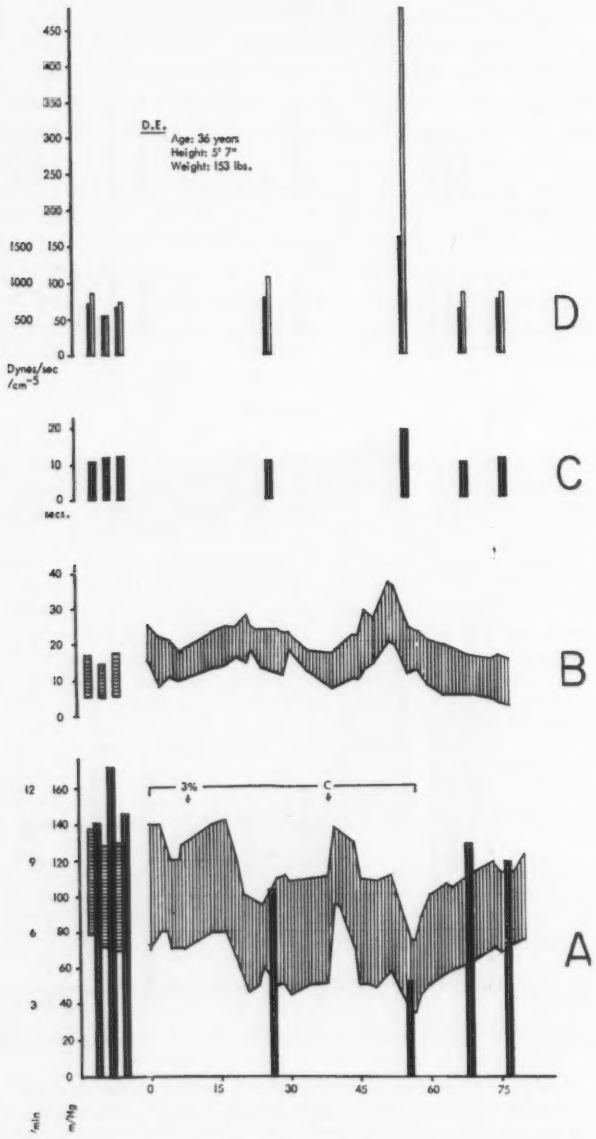

FIGURE 2. Same as Figure 1.

L./min. The vein-to-artery circulation time was unchanged at 11 seconds, the total peripheral resistance had risen slightly and the total pulmonary resistance markedly.

There was some technical difficulty while the change to a closed system was being made, so that anaesthesia lightened rapidly and the blood pressure rose to 135/95 mm. Hg; meanwhile the pulmonary artery pressure had steadily fallen to pre-induction levels. Following this, the systemic blood pressure fell slowly and steadily with a concomitant rise in pulmonary artery pressure. Respiration was controlled. When a systolic pressure of 75 mm. Hg had been reached, the cardiac output had fallen to 3.9 L/min., and the pulmonary artery pressure to 23/13 mm. Hg. The vein-to-artery circulation time had increased to 19 seconds. There was a substantial increase of total peripheral resistance, but the most striking feature was the great rise of total pulmonary resistance. A nodal rhythm was observed as the systemic blood pressure reached its nadir.

After halothane had been discontinued, the systemic blood pressure rose gradually to 120/75 mm. Hg, and the pulmonary pressure fell to 15/2 mm. Hg. The cardiac output was measured twice during the recovery period and found to have risen to the control level. Circulation time and systemic and pulmonary resistances were also back to normal.

Subject III (Fig. 3)

Induction was as previously described, the 3 per cent concentration being reached in 5 minutes. The subject was moaning and struggling a little during this time, and the electrocardiogram showed some ventricular extrasystoles. As anaesthesia deepened, these disappeared and the blood pressure fell rapidly from 160 mm. Hg to a little over 100 mm. Hg systolic, the pulmonary artery pressure rose briefly from 23/9 mm. Hg to 32/15 mm. Hg and then stabilized at 25 mm. Hg systolic, and the cardiac output fell from 12 L./min. to 7.8 L./min. There was no significant change in the vein-to-artery circulation time, but the total peripheral resistance increased slightly and the total pulmonary resistance markedly. As soon as the systemic blood pressure had reached a stable level, a nodal rhythm, alternating with an inverted T wave, was observed in Lead II of the electrocardiogram. These abnormalities persisted on and off throughout the experiment.

When the circulatory parameters had been stable for 10 minutes, the anaesthetic system was closed and respiration was first assisted and then controlled. The systemic blood pressure now fell abruptly to 24/16 mm. Hg, at which level halothane administration was stopped. The S-T segment in Lead II of the electrocardiogram was now depressed, the pulse rate was 48/min., the pulmonary artery pressure was 15/7 mm. Hg, and the cardiac output had fallen to 5.8 L./min. The vein-to-artery circulation time had increased from 13 seconds to 16½ seconds. The total peripheral resistance had fallen to pre-anaesthetic levels, but a further marked rise in the total pulmonary resistance had occurred.

After halothane had been discontinued, the systemic blood pressure rose spontaneously. Within 10 minutes it had reached 90/50 mm. Hg and the pulmonary artery pressure had returned to 20/5 mm. Hg. The cardiac output was now 8.2 L./min., the vein-to-artery circulation time had returned to normal, the peripheral resistance had increased somewhat and the total pulmonary resistance had fallen. Nodal rhythm was again noted intermittently.

Lanatoside C 0.4 mg. was administered intravenously after the output determination had been completed. This seemed to have no immediate effect on the course of events, other than that the nodal rhythm disappeared.

Twenty minutes later the systolic blood pressure had risen to 140/80 mm. Hg and the output had reached pre-induction levels.

Subject IV (Fig. 4)

Anaesthesia was induced in the usual manner and after some struggling and coughing a concentration of 3 per cent halothane was reached after 17 minutes. The systemic

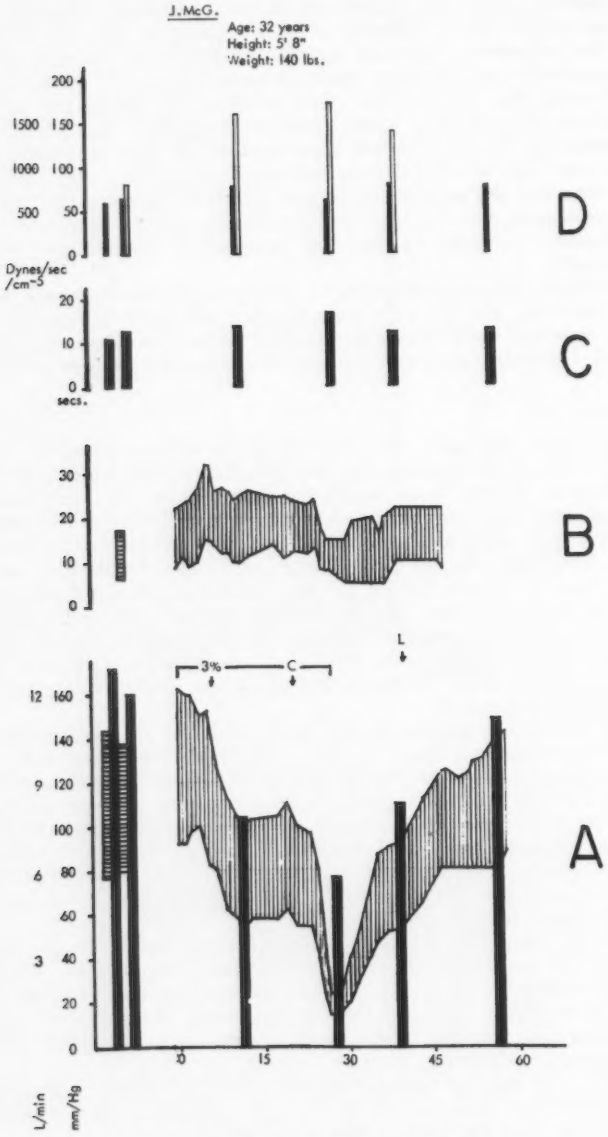

FIGURE 3. Same as Figure 1. L, Lanatoside C given.

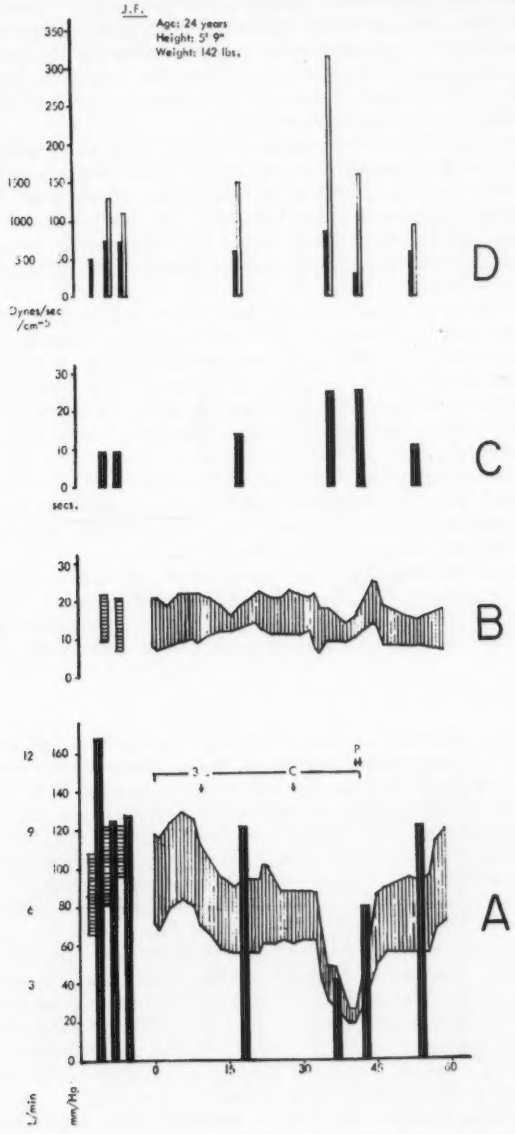

FIGURE 4. Same as Figure 1. P, phenylephrine given.

blood pressure rose from 120/70 mm. Hg to 130/80 mm. Hg during this rather stormy induction, but the pulmonary artery pressure was unchanged.

Following this, the systemic blood pressure fell to, and became stable at, 90/55 mm. Hg. systolic and the pulmonary artery pressure fell to 16/12 mm. Hg from 22/10 mm. Hg. The cardiac output was unchanged, but the vein-to-artery circulation time increased from 9 to 14 seconds. The total peripheral resistance fell and the total pulmonary resistance was markedly increased.

When the blood pressure had been stable for a little over 15 minutes, the anaesthetic system was closed and respirations were controlled. Nodal rhythm soon appeared, and after 3 minutes the systemic pressure fell steeply to 48/30 mm. Hg. There were occasional ventricular extrasystoles and the pulse rate had declined to 55/min. The cardiac output now had fallen to 3.1 L./min., the vein-to-artery circulation time was 24 seconds, the total peripheral resistance was increased a little and the total pulmonary resistance had risen greatly.

Meanwhile, the systemic blood pressure had fallen still further to 25/20 mm. Hg and the pulmonary artery pressure to 15/9 mm. Hg. The administration of phenylephrine 0.25 mg. intravenously, followed 3 minutes later by another 0.5 mg., raised the systemic blood pressure to 85/45 mm. Hg. Administration of halothane was now discontinued. Immediately following this, the cardiac output was found to have risen to 6 L./min. A nodal rhythm persisted although ventricular extrasystoles were seen no longer. The pulmonary artery pressure had risen to 16/10 mm. Hg, from a low of 13/9 mm. Hg at the time of greatest systemic hypotension. The vein-to-artery circulation time was unchanged, but the peripheral resistance was markedly diminished and the total pulmonary resistance, although much reduced from the previous reading, was still significantly elevated above the controls.

During the next 10 minutes, the blood pressure rose steadily to 99/55 mm. Hg, the nodal rhythm disappeared, and the pulmonary artery pressure declined gradually to 15/8 mm. Hg. At the end of this time the cardiac output was 9.1 L./min., the circulation time was normal and the peripheral and pulmonary resistances more nearly normal.

Subject V (*Fig. 5*)

This was a smooth induction, a concentration of 3 per cent halothane being attained within 5½ minutes. As anaesthesia progressed, the systemic blood pressure fell steeply from 130/85 mm. Hg to 62/30 mm. Hg and then rose gradually to stabilize at 70/40 mm. Hg; the pulmonary artery pressure declined from 22/11 mm. Hg to 13/5 mm. Hg immediately after induction; the pulse rate from 60/min. to 48/min. When the circulatory parameters were stable at these levels, the cardiac output was found to be 5.9 L./min. and the vein-to-artery circulation was unchanged at 23 seconds, but the total peripheral resistance had fallen slightly and the total pulmonary resistance moderately.

Soon after the cardiac output measurement had been done, the subject's pulse rate fell to 36/min. The injection of atropine 0.4 mg. intravenously raised the rate to 68/min. within 1 minute and caused the systemic blood pressure to rise to 100/60 mm. Hg. The pulmonary artery pressure rose to 19/6 mm. Hg. At this time the cardiac output had increased to 7.8 L./min., and the vein-to-artery circulation time had decreased to 11 seconds. The total peripheral resistance had risen slightly and the total pulmonary resistance had fallen a little.

Halothane was then administered from a modified Boyle bottle with the lever at 10, the oxygen flow being unchanged. Respiration was assisted and later controlled. There followed a sharp drop of systemic blood pressure, which reached 40/25 mm. Hg after 7 minutes, accompanied by a slower fall of pulmonary artery pressure to 10/2 mm. Hg. The cardiac output at this time had fallen to 6.2 L./min., the vein-to-artery circulation time had increased to 22 seconds and both peripheral and pulmonary resistances had fallen further.

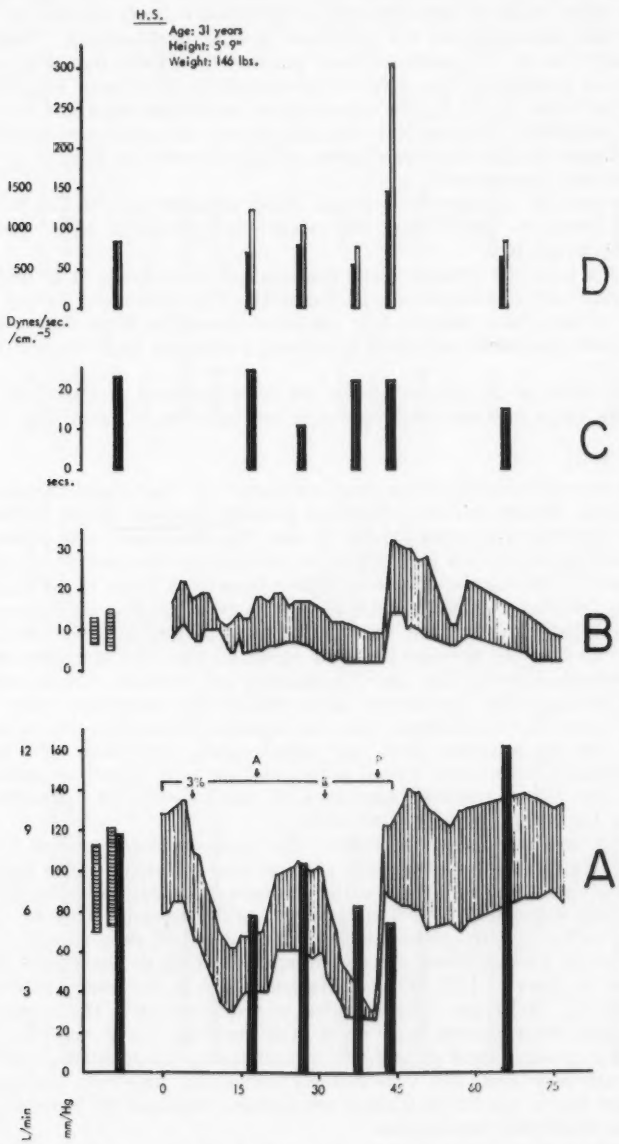

FIGURE 5. Same as Figure 1. B, change from Fluotec to Boyle vaporizer; A, atropine sulphate 0.4 mg. given; P, phenylephrine given.

Two minutes after these determinations had been completed the systemic blood pressure had fallen to 30/25 mm. Hg and the pulmonary artery pressure to 9/2 mm. Hg. At this time phenylephrine 2.5 mg, was injected intravenously. There was an immediate sharp rise of the systemic blood pressure to 122/86 mm. Hg and of the pulmonary artery pressure, after a delay of 30 seconds, to 33/14 mm. Hg. The cardiac output now had fallen to 5.5 L.; the vein-to-artery circulation time had not changed, but the total peripheral resistance had risen sharply and the total pulmonary resistance greatly. The P wave on the electrocardiogram became inverted in Lead II at this time. Halothane was now discontinued.

During the next 10 minutes the systemic blood pressure rose to 140/80 mm. Hg and then fell slowly to 120/75 mm. Hg, while the pulmonary artery pressure fell gradually to 11/6 mm. Hg.

Seven minutes later, the systemic blood pressure had risen slowly to 130/80 mm. Hg and the pulmonary artery pressure was 16/5 mm. Hg. The cardiac output now had risen to over 12 L./min., the vein-to-artery pressure circulation time had returned to normal, and both peripheral and total pulmonary resistances had returned to control levels.

Ten minutes later, as the subject awoke, the systemic blood pressure had remained unchanged, but the pulmonary artery pressure had fallen to 8/2 mm. Hg.

Subject VI (Fig. 6)

This was a smooth induction in the usual manner; 3 per cent halothane was attained within 4 minutes. During this time the blood pressure dropped steeply to 64/40 mm. Hg and then gradually rose again to 80/45 mm. Hg. Pulmonary artery pressure was essentially unchanged at 17/5 mm. Hg after it had risen momentarily to 20/4 mm. Hg after induction. The cardiac output had fallen from 9.3 L./min. to 8.4 L./min. with an increase in the vein-to-artery circulation time from 12 seconds to 15 seconds. The total peripheral resistance fell, but the total pulmonary resistance was unchanged.

When the systolic blood pressure had risen to 85/60 mm. Hg, atropine 0.4 mg. was given intravenously. During the next 15 minutes the systemic blood pressure fell slowly to 60/45 mm. Hg. During the same period the pulmonary artery pressure averaged 15/8 mm. Hg. Two minutes after the injection of atropine, the cardiac output had risen to its pre-induction level, the vein-to-artery circulation time and total peripheral resistance had risen to control values, and the total pulmonary resistance had not changed. The fall of systemic blood pressure was followed by a spontaneous rise to 80/55 mm. Hg within the next 5 minutes.

At this point, rebreathing was instituted. The systemic blood pressure fell rapidly to 48/40 mm. Hg. The pulmonary artery pressure rose at first to 22/14 mm. Hg and then fell to 11/7 mm. Hg. At this time the cardiac output had fallen to 5.7 L./min., the vein-to-artery circulation time had increased to 15 seconds, the total peripheral resistance had fallen, and the total pulmonary resistance had risen steeply. The intravenous injection of phenylephrine 2.5 mg. was followed by an immediate rise of the systemic blood pressure to 125/105 mm. Hg and a rise of pulmonary artery pressure to 38/22 mm. Hg. Halothane administration was discontinued. Three minutes later a cardiac output determination was done. The resultant curve was flat and prolonged and did not lend itself to accurate measurements. Undoubtedly, however, the output was extremely low. The vein-to-artery circulation time was increased to 20 seconds. There was a run of ventricular extrasystoles followed by sinus rhythm and return of more ventricular extrasystoles.

Both systemic and pulmonary pressures returned to pre-anaesthetic levels during the next 30 minutes. Cardiac outputs, measured on two occasions, had returned to normal levels, as had the vein-to-artery circulation time and the total peripheral resistance. The total pulmonary resistance remained elevated at the first reading but had returned to pre-anaesthetic levels on the second.

Just before the patient wakened, his systemic blood pressure was 115/80 mm. Hg and his pulmonary pressure 12/6 mm. Hg; all other parameters were normal.

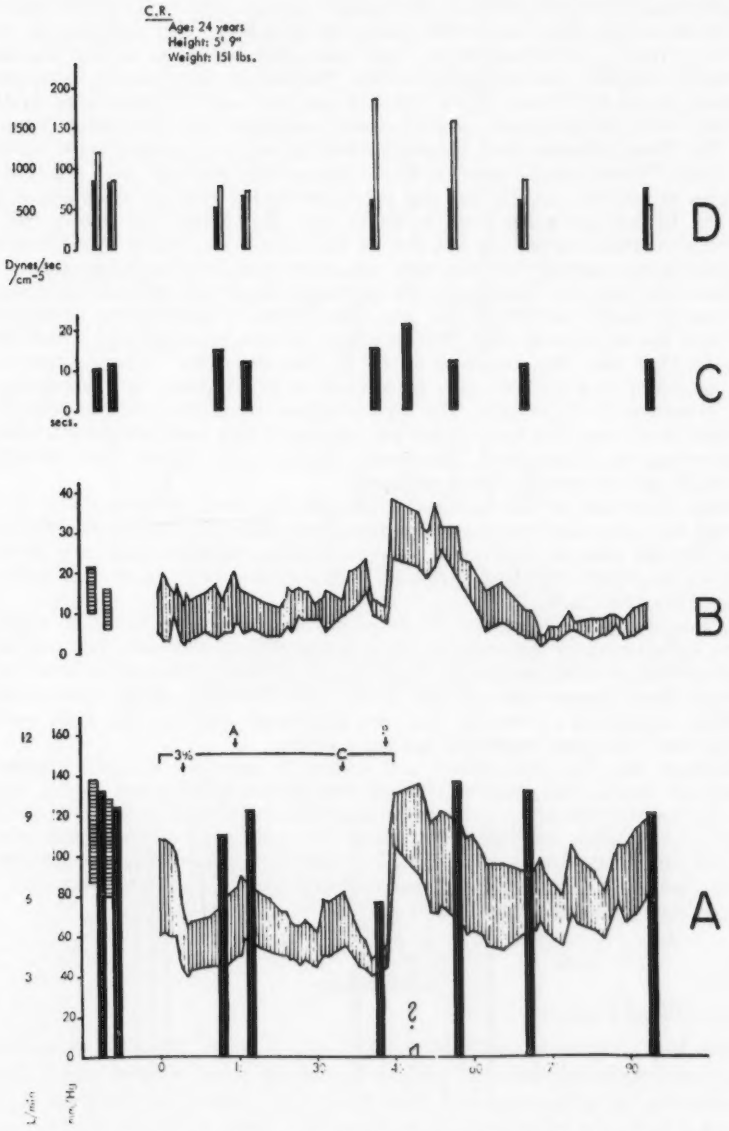

FIGURE 6. Same as Figure 1. A, atropine given; P, phenylephrine given.

Subject VII (Fig. 7)

Anaesthesia was administered in the routine manner, but the induction was somewhat more stormy than usual with much breath-holding and coughing; it took 9 minutes to reach a concentration of 3 per cent. Even then, the subject was holding his breath, coughing, and struggling a little. Meanwhile the systemic blood pressure had risen from 145/85 mm. Hg to 160/110 mm. Hg and had then fallen to 130/80 mm. Hg, while the pulmonary artery pressure had risen from 16/7 mm. Hg to 20/8 mm. Hg. Three minutes later succinylcholine 10 mg. was administered to smooth anaesthesia. Thereafter, the systemic blood pressure fell gradually until it had reached a plateau at 105/64 mm. Hg and the pulmonary artery pressure stabilized at 20/10 mm. Hg, having previously risen to 25/11 mm. Hg. When the plateau had been reached, the cardiac output was found to be 10 L./min. The total peripheral resistance had diminished somewhat and the total pulmonary resistance had increased.

Rebreathing was now instituted with controlled respiration and the halothane concentration gradually reduced to 0.5 per cent. Within 7 minutes the blood pressure had fallen to 60/40 mm. Hg. The pulmonary artery pressure, which had initially fallen to 17/7 mm. Hg, returned to 20/12 mm. Hg. The cardiac output at this time had fallen to 4 L./min. with an increase in the vein-to-artery circulation time from 10 seconds to 13 seconds. The total peripheral resistance had increased to pre-induction levels and the total pulmonary resistance had risen markedly. While the cardiac output was being done, anaesthesia became a little lighter. This was reflected by a slight rise of systemic blood pressure.

Further deepening of the anaesthesia brought the blood pressure down to 40/30 mm. Hg, the pulmonary artery pressure remaining essentially unchanged. The cardiac output was the same as before but the vein-to-artery circulation time was much prolonged at 19 seconds, the total peripheral resistance was reduced, and the pulmonary resistance remained high.

The intravenous administration of phenylephrine 2.5 mg. was followed, within one minute, by a return of the systemic blood pressure to 130/90 mm. Hg, and a steep rise of pulmonary artery pressure to 41/21 mm. Hg. The cardiac output failed to keep pace with these changes and was only 5.7 L./min. The vein-to-artery circulation time had fallen slightly to 17 seconds, the total peripheral resistance had risen markedly, and the total pulmonary resistance had risen further.

Halothane was now discontinued and within 10 minutes the cardiac output had returned to 10.2 L./min., neither systemic nor pulmonary pressures having changed. The vein-to-artery circulation time was now normal at 10 seconds, the total peripheral resistance had fallen and the total pulmonary resistance, although still markedly elevated, had returned to more acceptable levels. The pulmonary artery pressure was still elevated at 42/11 mm. Hg and remained high until the subject awoke.

No cardiac arrhythmias were noted during the experiment.

Discussion

Systemic Blood Pressure

There is a progressive, sometimes rapid, fall of blood pressure as anaesthesia is deepened with halothane. A plateau is established at a level which depends upon the concentration employed. Sometimes this plateau is established at a level somewhat higher than that reached during the initial fall, probably because of compensatory readjustments in the peripheral vascular bed.

If the concentration is further increased, for example, if rebreathing is permitted, the pressure falls further. Often this fall begins after a very short period of time, and may be precipitous in the presence of controlled respiration. This

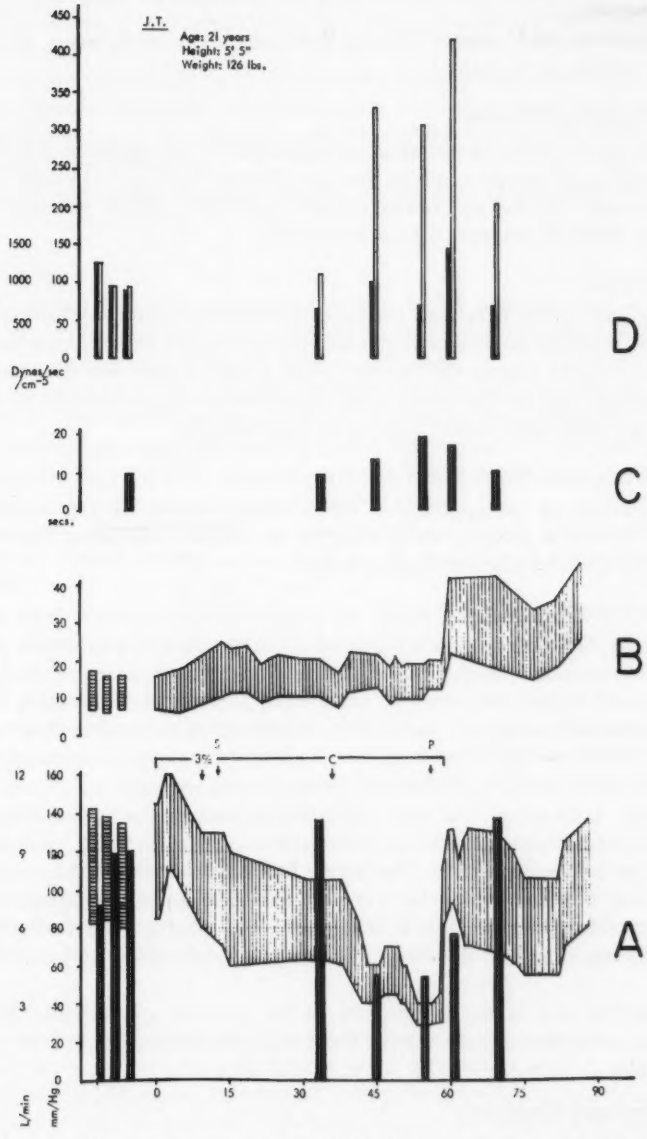

FIGURE 7. Same as Figure 1. S, succinylcholine 10 mg. given; P, phenylephrine given.

second fall of pressure, due to a deliberate overdose, results in marked narrowing of the pulse pressure.

The blood pressure quickly rises to pre-anaesthetic levels when the administration of halothane is stopped.

Pulmonary Artery Pressure

There is usually a slight elevation of pulmonary artery pressure, which persists until the systemic pressure falls to very low levels. This fall is usually associated with a concomitant fall of pulmonary artery pressure which remains low even though the systemic pressure returns to normal.

Cardiac Output

The cardiac output falls with the systemic blood pressure until the plateau of pressure is reached. If further hypotension is produced by an overdose of halothane, the cardiac output falls to the point where it becomes inadequate. The cardiac output rises *pari passu* with the spontaneous rise of blood pressure which occurs after halothane administration is discontinued.

Vein-to-Artery Circulation Time

A comparison of the appearance times of the various cardiac output curves indicates clearly a progressive slowing of circulation related to the degree of hypotension and reduction of cardiac output.

Total Peripheral Resistance

This is the sum of the pressure-flow relationships in all parts of the peripheral circulation. A change in the total peripheral resistance indicates that vasoconstriction or dilatation in one area or areas exceeds the opposite reaction in another field. It does not necessarily mean that vasoconstriction or dilatation is general; nor does it indicate which parts of the vascular tree are predominantly affected.

The peripheral resistance declines as the blood pressure falls and settles to the plateau, indicating that this moderate hypotension may be the result of peripheral vasodilatation. The presence, in some subjects, of a definite skin blush supports this conclusion. The severe hypotension produced by an overdose of halothane is accompanied by a substantial increase of peripheral resistance. This presumably is a defensive reaction on the part of the vascular system, tending to maintain circulation in the face of hypotension and a low cardiac output.

Towards the end of the experiment, as the level of anaesthesia lightens, the peripheral resistance, together with the other parameters, returns to pre-anaesthetic levels.

Total Pulmonary Resistance

Total pulmonary resistance invariably increases, often quite sharply, as the plateau of hypotension is reached. Total pulmonary resistance is the sum of the resistance across the pulmonary arterioles and the resistance of the left heart to the inflow of blood from the pulmonary circulation. It is a matter of speculation whether this increased total pulmonary resistance is due to an increased

pulmonary arteriolar constriction, as a direct effect of halothane, or to an elevated left ventricular diastolic pressure associated with the diminished output of the left heart.

As the cardiac output falls with an overdose of halothane, the total pulmonary resistance increases markedly, and may reach very high levels. This increase is almost certainly due to the resistance offered by the left heart, which is unable to propel the blood offered to it.

Total pulmonary artery resistance returns to pre-anaesthetic levels as halothane is eliminated from the body.

Effect of Drugs

Phenylephrine was administered when the blood pressure had reached its lowest levels in four of the seven subjects. Large doses of phenylephrine raised the systemic pressure immediately to pre-anaesthetic levels, and the pulmonary artery pressure to very high levels. Smaller doses caused a more gradual rise in Subject IV. The cardiac output either fell further or was unchanged, indicating that the myocardium was sufficiently affected by halothane to be unable to cope with the increased peripheral resistance against which it had to work. Ventricular extrasystoles were seen in two subjects at this time.

The cardiac output returned to pre-anaesthetic levels and severe cardiac irregularities ceased within a few minutes of stopping the administration of halothane.

Atropine sulphate, given twice during the initial fall of systemic blood pressure towards the plateau, caused reversal of the hypotension for a period of time in one subject in whom it also helped to raise the pulmonary artery pressure. Atropine reversed bradycardia promptly. The cardiac output increased and vein-to-artery circulation time shortened. In both subjects following administration of atropine, total peripheral resistance increased, but total pulmonary resistance was not significantly affected. These observations raise some interesting problems as to the action of atropine.

Lanatoside C given only once during the spontaneous return of pressure following hypotension seemed to have little if any effect, other than that of abolishing a nodal rhythm. Whether this was due to the drug or merely a coincidence remains a matter of speculation, since nodal rhythm tended to disappear spontaneously in other subjects as the level of anaesthesia became lighter.

Cardiac Rhythm

The electrocardiogram (Lead II) was unchanged in two subjects.

The P wave inverted in one subject soon after induction; a nodal rhythm appeared when the plateau of systemic blood pressure was reached. Nodal rhythm was present in two others when the systemic pressure reached its nadir and in one the P wave inverted after the administration of phenylephrine.

Ventricular extrasystoles occurred in three subjects: once early in the experiment, once as the systemic blood pressure reached its lowest level, and once after phenylephrine. The S-T segment was depressed in one subject at the time of maximum hypotension.

Cardiac Rate

Bradycardia invariably occurred during halothane anaesthesia. It could be reversed by the use of atropine. If it was permitted to persist, the bradycardia often culminated in nodal rhythm.

Electroencephalogram

There was no correlation between the apparent depth of anaesthesia and the electroencephalographic pattern. Even when marked hypotension was produced, the electroencephalogram gave the appearance of relatively light levels of surgical anaesthesia. Presumably then, severe cardiovascular depression occurs in light planes of anaesthesia long before anaesthesia is deep enough to cause burst suppression.

Laboratory Investigations

Laboratory investigations were carried out not only to check the effects of halothane following an acute experiment, but also to exclude any harmful effects on parenchymatous function of repeated dye injections.

The only consistent finding was a mild leucocytosis on the day following the experiment. This was no doubt due to the trauma inflicted and the administration of drugs. All subjects showed a slight increase of haemoglobin and packed cell volume on the day following the experiment. Probably this was due to dehydration since they had only had one meal in 30 hours.

One subject who had had a pre-anaesthetic elevation of gamma globulin, thymol turbidity, and zinc sulphate turbidity had a further rise of zinc sulphate turbidity on the day following the experiment and at that time had also a double plus thymol flocculation test. His liver function tests, repeated six days later, had returned to pre-experimental levels.

The urinary tests always gave essentially normal results.

Neither administration of large doses of halothane for periods up to one hour, nor the repeated injection of Cardio-Green, exerted any deleterious effect on parenchymatous functions in these seven subjects.

Summary and Conclusions

The administration of controlled concentrations of halothane lowers the systemic blood pressure. This hypotension tends to reach a plateau and does not seriously affect cardio-vascular efficiency. There is good evidence that this hypotension is due to peripheral vasodilatation; it is associated with increased total pulmonary resistance.

Overdoses of halothane can easily be administered if either a closed system or a poorly calibrated vaporizer is used. The hypotension thus produced may reach profound levels in a very short period of time and seriously impairs circulatory efficiency. It is associated with some peripheral vasoconstriction. The increased total pulmonary resistance indicates that the hypotension is most likely due to myocardial impairment.

Bradycardia invariably occurs and becomes pronounced when excessive doses of halothane are administered. It can be reversed by atropine sulphate, which may also raise the blood pressure from its early plateau.

The profound hypotension produced by overdoses of halothane reverts spontaneously when the administration of the drug is stopped. It is imperative that the hypotension be recognized early because of the severe effect it has on myocardial efficiency. The hypotension can also be reversed by vasopressors, but these must be used cautiously since they may produce ventricular extrasystoles. The most frequent electrocardiographic abnormalities produced by halothane are inversion of the P wave and nodal rhythm. Ventricular extrasystoles may occur early or when hypotension becomes extreme.

Halothane does not seriously affect the circulation provided its concentration it rigorously controlled by means of a suitable vaporizer. Its administration in a closed system or from unsuitable vaporizers is fraught with grave potential hazard. The potentially adverse effects of halothane upon the cardiovascular system are such that the use of this drug must be restricted to those familiar with its properties.

Acknowledgments

The authors wish to express their thanks to Dr. G. Bray, Mr. K. Varley, and Miss B. Cox, all of the Cardio-pulmonary Laboratory, for technical assistance, and to Mr. K. Drummond of the Department of Anaesthesia for assistance with the measurements of the blood pressure changes.

Résumé

Nous avons fait des expériences sur des volontaires du sexe masculin dans le but d'étudier les effets de l'halothane sur le système cardiovasculaire.

Nous avons mesuré la pression artérielle périphérique directement au moyen d'une aiguille à demeure dans une artère; nous avons mesuré la pression artérielle pulmonaire au moyen d'un cathéter pour le cœur. Nous avons déterminé le débit cardiaque un certain nombre de fois durant chaque expérience au moyen d'une méthode de dilution en employant une nouvelle teinture verte—Cardio-vert®—comme indicateur; nous avons fait l'échantillonnage artériel au moyen d'un oxymètre cuvette. Des données obtenues, nous avons calculé le temps de circulation veine à artère, la résistance totale périphérique et la résistance pulmonaire totale.

Continuellement, nous avions comme contrôle: l'électroencéphalogramme et un électrocardiogramme en deuxième dérivation.

Avant et après les expériences, nous avons fait subir des tests hépatiques et rénaux.

Nous avons constaté que l'administration d'halothane à des concentrations contrôlées fait baisser la pression sanguine systémique. Cette hypotension a tendance à atteindre un plateau et n'affect pas sérieusement l'efficacité du système cardiovasculaire. Nous avons raison de croire que cette hypotension est occasionnée par une vasodilatation périphérique; elle s'accompagne d'une augmentation de la résistance pulmonaire totale.

Nous avons constaté aussi qu'il est facile de faire du surdosage avec l'halothane, soit en employant un circuit fermé, soit en employant un vaporisateur dont la calibration n'est pas précise. En ces circonstances, l'hypotension constatée peut

atteindre très rapidement des niveaux très bas et réduire sérieusement l'efficacité de la circulation. Elle s'accompagne d'une certaine vasoconstriction périphérique. L'augmentation de la résistance pulmonaire totale nous porte à croire que l'hypotension observée serait attribuable à des troubles myocardiques.

Nous avons noté l'apparition constante d'une bradycardie s'accentuant quand les doses d'halothane augmentent. On peut faire disparaître cette bradycardie en administrant de l'atropine ce qui a pour effet également d'élever la pression artérielle à son plateau initial.

L'hypotension marquée produite par des surdosages d'halothane disparaît spontanément si l'on cesse l'administration du médicament. Il s'impose de dépister précocement l'hypotension à cause des effets sérieux qu'elle produit sur l'efficacité myocardique. On peut corriger l'hypotension en employant également des vasopresseurs, mais il faut être prudent en les employant, car ils peuvent provoquer des extrasystoles. Les anomalies électrocardiographiques les plus fréquemment observées ont été une inversion de l'onde P et un rythme nodal. Nous avons observé que des extrasystoles ventriculaires peuvent apparaître précocement ou encore quand l'hypotension devient très marquée.

Pourvu que sa concentration soit contrôlée de façon rigoureuse au moyen d'un vaporisateur approprié, l'halothane n'affecte pas sérieusement la circulation. Si on l'administre en circuit fermé ou au moyen d'un vaporisateur inadéquat, l'halothane peut constituer un risque sérieux. L'halothane peut avoir sur le systéme cardiovasculaire des effets tellement nuisibles que son emploi devrait être réservé à ceux qui connaissent bien ses propriétés.

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Errata

Sodium Methitural: A Clinical Study, by Gordon M. Wyant *et al.*, this Journal, Volume 5, page 265 (July, 1958), Table V: the following should be the times for the mean duration of operation.

	Mean
Thiopental c	12 min. 40 sec.
Methitural c	9 min. 41 sec.
Methitural s	8 min. 45 sec.
Methitural (exp.) s	10 min. 31 sec.

FLUOTHANE-ETHER: AN AZEOTROPIC MIXTURE[1]

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FLUOTHANE,[5] a volatile anaesthetic agent, is 1,1,1-tri-fluoro-2,2-bromochloroethane. It is non-inflammable and non-explosive (1). For the maintenance of anaesthesia, a concentration of 0.5 to 1.5 per cent is sufficient (2).

Induction is easy, rapid, with absence of coughing, vomiting, and salivary and bronchial secretions; surgical anaesthesia is present within 3 to 4 minutes. The changes in the level of anaesthesia occur rapidly so that a calibrated vaporizer is very useful to regulate the concentration. The recovery stage is calm and complete in 3 to 15 minutes following anaesthesia.

On the other hand, Fluothane is only slightly analgesic before the loss of conjunctival reflex; it decreases the depth of respiration and depresses blood circulation (3). This results probably from a central depression and a decrease of the adrenalo-sympathetic response. Lastly, constant care is needed in Fluothane anaesthesia.

To provide an adequate oxygenation and a sufficient ventilation, oxygen should be given in excess, respiration should be assisted if necessary and only a vaporizer with accurate calibration should be used in order to prevent too deep a level of anaesthesia. If Fluothane anaesthesia is not well conducted, that is to say, if induction is too rapid and anaesthesia is too deep, a vagal hyperstimulation of the heart and a too rapid anaesthesia saturation of the cardiac muscle will lead to bradycardia which could result in cardiac arrest (3). Cardiac arrhythmias are not frequent; they seem to be related to a disturbance in the respiratory exchange, to Fluothane itself, and to the level of anaesthesia. These arrhythmias will decrease gradually with the experience and the skill of the anaesthetist. Following induction, a decrease of 20 to 50 mm. of mercury in the blood pressure is seen in most patients; later on, the blood pressure will vary with the neuro-vegetative status of the patient, with the lowering of blood volume, with the concentration of Fluothane in blood, with the position of the patient, the degree of hyperventilation and of positive pressure.

In securing Fluothane, an anaesthetic agent which offers such advantages as central sedation, potency, non-explosive atmosphere, and possibility of open-drop method, we searched for ways to raise its margin of safety.

The association of volatile anaesthetic agents is well known, and the idea of mixing di-ethyl ether with Fluothane has been put forward for the following reasons: generally, anaesthetic mixtures are made with agents of different boiling points and of varied pharmacological properties but of successive actions; quali-

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ties which often constitute a clinical disadvantage. However, in certain conditions, azeotropic mixtures result, that is, solutions with common boiling points, producing a concomitant but not successive action. In these circumstances, the proportions of the mixture remain constant. Yet, we have not heard of any azeotropic mixtures demonstrated in anaesthesia. In the hope that we might obtain a successful association and an azeotropic mixture, one part di-ethyl ether was mixed with two parts Fluothane for laboratory and clinical studies on August 27, 1957.

Our researches were carried out on toxicity, degree of inflammability, and on identification of an azeotropic mixture; the results obtained are reported in the complementary paper.

By a fortunate accident we were within 2 per cent of the azeotropic mixture likely to produce surgical anaesthesia with less than 1 per cent di-ethyl ether and 2 per cent Fluothane in the breathed gases. This azeotropic mixture boils at a constant boiling point and provides, in the vapour phase, a respirable gaseous anaesthetic mixture wherein the two components are present in essentially the same relative proportions as in the liquid which was volatilized.

The vapour of expiration was condensed by passing it through a trap cooled by means of a cooling mixture of acetone and dry ice; the composition of the condensate is identical with the liquid which was vaporized. This mixture is as stable on storage as Fluothane itself; it is non-inflammable in clinical use. The lower limit of inflammability with oxygen is 10.9 per cent.

This discovery led us to investigate other mixtures, such as Chloroform-di-ethyl ether and Vinethene-di-ethyl ether, which, we found, give azeotropic compounds, but in proportions different from those already used in clinical anaesthesia.

The advantages of di-ethyl ether are well known (4). Di-ethyl ether decreases the intracardiac conductibility. If a displacement of the pacemaker is produced, it is not generally followed by extrasystoles; this can antagonize the increase of the myocardial excitability caused by Fluothane. At a low concentration, di-ethyl ether produces sympathetic stimulation that may partly counterbalance the hypotensive effect of Fluothane. By reflex action, di-ethyl ether increases the secretion of adrenalin and of noradrenalin.

Blood pressure, cardiac rhythm, and cardiac output show an initial elevation which augments to the point where 100 cc. of blood contain 91 to 110 mg. of di-ethyl ether (5). Cardiac output increases up to the second plane of the third stage. At this level, it is 241 per cent higher than normal (12).

At the second plane of the surgical stage, blood contains 120 mg. of di-ethyl ether in 100 cc. of arterial blood (5). Knowing that with a concentration of 25 mg. of di-ethyl ether in 100 cc. of arterial blood the respiratory volume augments, one can expect an improvement of the respiration (5). This concentration of di-ethyl ether is equal to that employed by Artusio to obtain analgesia is cardiac surgery, that is, 25 to 50 mg, of di-ethyl ether in 100 cc. of arterial blood (6). Thus mixed with Fluothane, di-ethyl ether may augment analgesia, increase ventilation, and diminish pain reflexes and reflex arrhythmia during a too superficial anaesthesia (Table I).

TABLE I

PERCENTAGE CONCENTRATION OF FLUOTHANE AND ETHER IN THE INSPIRED MIXTURE AND IN ARTERIAL BLOOD

Anaesthetic	Inspiration	Fluothane	Ether	Stage III Arterial blood (mg.) (hypothetical)
Fluothane	3%	3%	0%	15 mg.
Fluothane–ether	3%	2.04%	0.96%	Fluothane: 10 to 12 mg. Ether: 12 to 40 mg.

The boiling point of Fluothane-ether is 51.5°C. compared with 50.2°C. for Fluothane and with 36.5°C. for di-ethyl ether; this makes the "Fluotec" vaporizer valuable in indicating the percentage of the mixture being delivered. The proportions of azeotropic mixture are 68.3 of Fluothane and 31.7 of di-ethyl ether for 100 cc. of the mixture (Table II).

TABLE II

BOILING POINTS OF FLUOTHANE, ETHER, AND THE AZEOTROPIC MIXTURE

Anaesthetics	Boiling point
Fluothane	50.2° C.
Ether	36.5° C.
Fluothane–ether 68.3 cc. + 31.7 cc. = 100 cc.	51.5° C.

With the "Fluotec" vaporizer, a surgical anaesthesia is obtained at the 2.5 to 3 per cent mark and maintenance is around 1.5 per cent; this gives in the inspired volume approximately 0.4 per cent to 0.95 per cent di-ethyl ether per volume and 0.8 per cent to 2 per cent Fluothane. It should be noted that 120 to 150 mg. of di-ethyl ether per 100 cc. of blood are obtained after inhalation of 2 to 4 per cent di-ethyl ether in the breathed air (5). A percentage of 0.4 to 0.95 di-ethyl ether per volume in inspired air could give, after several minutes, a concentration of 12 to 40 mg. in 100 cc. of arterial blood.

The lower limit of inflammability of di-ethyl ether with oxygen is 2.1 per cent this is a concentration much greater than the percentage of the mixture under study, if we consider that the vapour phase of inspiration and of expiration remains in the same proportion as the liquid composition.

Methods used included Boyle apparatus with or without "Fluotec" vaporizer, with open, semi-closed, or closed circuit; Heidbrink apparatus in semi-closed or closed circuit with oxygen; and open mask drop with continuous administration of oxygen by nasopharyngeal catheter.

Clinical Results

The mixture of Fluothane-ether gives an induction, an anaesthesia, and a recovery quite similar to those observed with Fluothane alone: rapid and easy induction without secretions and early depression of pharyngeal and laryngeal reflexes. Following induction with thiobarbiturates, the inhalation of the azeotropic mixture presents no difficulty, but one must correct by manual assistance the respiratory depression which prevails to a certain degree; however, ventilation is better than with Fluothane alone. Minute volume is increased by 20 per cent. Studies of the minute volume with Fink's valve and rotameter and with the "Ohio minute volume meter" show that, with Fluothane-ether, the minute volume increases during induction and also during maintenance under light anaesthesia; in the second plane with intubation and with absence of surgical stimulus, minute volume is augmented by one litre.

Hypotension is not constant; sometimes it will be 20 mm. of mercury or more during too rapid an induction, but, during maintenance, it tends to return to nearly a normal level. Nevertheless, especially in semi-closed and closed circuit, minute volume depression and apnoea happen quickly when Fluothane-ether concentration is increased and occur generally with a gradual fall of blood pressure. However, at this stage, peripheral circulation is better than with Fluothane alone. In some patients, it is not a fall of blood pressure but arrhythmia which accompanies the respiratory depression. When this happens, one must close the vaporizer and assist the respiration until a lighter plane of anaesthesia is obtained. Controlled respiration must always be done after having decreased the percentage of the mixture; that is to say, the potential hazards, although diminished, remain the same.

Eye signs are a reliable guide during induction and maintenance: pupils may dilate during the second stage and contract at the third stage to remain pin-point at the third plane. Eyes may open in deep anaesthesia.

The conduct of anaesthesia is easier and the margin of safety is greater. Most of our patients (Table III) were anaesthetized under closed circuit with pure oxygen. Flaxedil was used in small doses to obtain relaxation during maintenance; blood pressure and pulse rate increase with this drug.

Many patients have been anaesthetized several times with the mixture; they had no apprehension with the repeated inductions and no signs of intoxication postoperatively. In the series with Fluothane alone, three cardiac arrests supervened with complete recovery to normal (7, 8). In the series with Fluothane-ether, no cardiac arrest took place. With the visoscope, under Fluothane-ether anaesthesia and in comparison to Fluothane anaesthesia alone, the electrical modifications are not diminished in the initial phase of the ECG, but are considerably minimized in the intermediate and final phase of the ECG tracing.

The analysis of the liquid in the vaporizing bottle of the circuit showed no decomposition after several days of use. On one occasion, the water accumulated in one bottle for one month showed a small fraction of free acids, but this was insignificant in practice (analysis suggested and made by Dr. Carl Von Seemann).

TABLE III

SUMMARY OF CASES ANAESTHETIZED WITH FLUOTHANE AND WITH THE FLUOTHANE-ETHER MIXTURE

Surgical procedures with Fluothane without ether			3,016
Surgical procedures with Fluothane + ether			
Minor		1,323	
Major		1,121	
			2,444
Techniques			
Fluothane + ether	1,052		
Fluothane + ether + Pentothal	687		
Fluothane + ether + Neraval	705		
Intubation			
No intubation	804		
Intubation without relaxant	616		
Intubation with Anectine	740		
Intubation with Flaxedil	284		
TOTAL			5,460

Samples of the vaporized mixture taken with a syringe will not ignite if a lighted match is introduced into the syringe; these samples were taken from the anaesthetic bag, from the circuit tubes and from the endotracheal tube during expiration.

On three occasions, during an anaesthesia of four-hour duration conducted with a one-way vale, the vapours of the expiration were collected and condensed in a glass flask refrigerated with acetone and dry ice. The composition of the condensed liquid was identical with the liquid which was vaporized. This recovered Fluothane-ether and the water of expiration contained no free acid.

The mixture of one part of Fluothane with one part of di-ethyl ether was experimented with in the open drop method and with an anaesthetic machine; induction is easy but slower, respiration is further improved, and there is no hypotension.

To sum up, this clinical and experimental observation, based on a total of 5,460 cases (7–11) of which 2,444 are with Fluothane-ether, enables us to foresee a more extended use of the Fluothane-ether azeotropic mixture.

RÉSUMÉ

Le mélange deux parties de Fluothane pour une partie d'éther fut étudié au point de vue de solution, toxicité, inflammabilité, puissance et réaction.

Les analyses de laboratoire nous ont démontré que le liquide de composition est azéotrope dans le voisinage de ces concentrations, de même que dans la phase gazeuse et dans la partie récupérée de l'expiration.

Au point de vue chimique, il est non-toxique et reste stable pendant plusieurs mois.

La limite inférieure d'inflammabilité est de 10.9% dans l'oxygène.

Au point de vue clinique, la fraction éther semble jouer un rôle stimulant sur la respiration et la circulation et augmenter ainsi la marge de sécurité. Si l'on augmente la concentration d'éther, l'effet est encore plus marqué.

L'expérience de 2,444 anesthésies administrées avec ce mélange nous porte à croire que cette méthode peut être utilisée avec avantages.

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PROPERTIES OF THE FLUOTHANE-ETHER ANAESTHETIC[1]

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PHYSICS AND CHEMISTRY may bring a happy contribution to medicine in the study and the choice of anaesthetics, as will be illustrated by the study of the characteristics of a new compound, made up of Fluothane and ether.

When Fluothane and ether were mixed together, heat was produced. The temperature rose to about 33°C. as Fluothane and ether passed into solution: this phenomenon is called heat of solution. This solution, which is produced with a decrease of volume due to the molecular attraction, is not an ideal solution.

In order to identify this mixture, the vapour pressure was determined and was found to be equal to 213.3 mm. Hg for a temperature of 24°C. while the vapour pressure of ether and Fluothane was respectively 442 mm. Hg and 264 mm. Hg for the same temperature. This showed us that the boiling point of the mixture should be slightly higher than that of Fluothane if an azeotropic compound had been discovered. An azeotropic compound is a mixture of two substances which distil at the same temperature.

By distilling a mixture composed of two parts of Fluothane and one part ether, the presence of an azeotropic compound was detected. This compound distils at a constant temperature of 51.5°C. in definite proportions: 31.7 per cent ether and 68.3 per cent Fluothane.

When distilling 75 cc. of such mixture, a first 5 cc. fraction boiling between 50.5°C. and 51.5°C. was recovered: this fraction was composed of 33 per cent ether and 67 per cent Fluothane. Then the temperature was raised to 51.5°C. and a second fraction of about 65 to 68 cc. of the azeotropic compound (31.7 per cent ether for 68.3 per cent Fluothane) were separated out. Finally, the temperature was lowered to 50.2°C. and a few drops of Fluothane were obtained.

When a mixture of 31.7 parts of ether and 68.3 parts Fluothane was boiled, it distilled at 51.5°C.: this was the boiling point of the mixture.

When such a mixture was vaporized in the Boyle apparatus by means of a current of 50 per cent nitrous oxide, vapours were condensed in a trap cooled by dry ice dissolved in acetone. As was expected, the mixture composition in the vapour phase remained the same as that in the liquid phase: 31.7 per cent ether and 68.3 per cent Fluothane. Analyses were made by determining densities by means of a precalibrated micropycnometer (Fig. 1).

Following these results other experiments were carried out. While a patient was being anaesthetized with this azeotropic mixture, the vapours produced by the expiration were condensed. The condensation products were water, ether, and Fluothane. After elimination of water, analyses proved that ether and Fluothane

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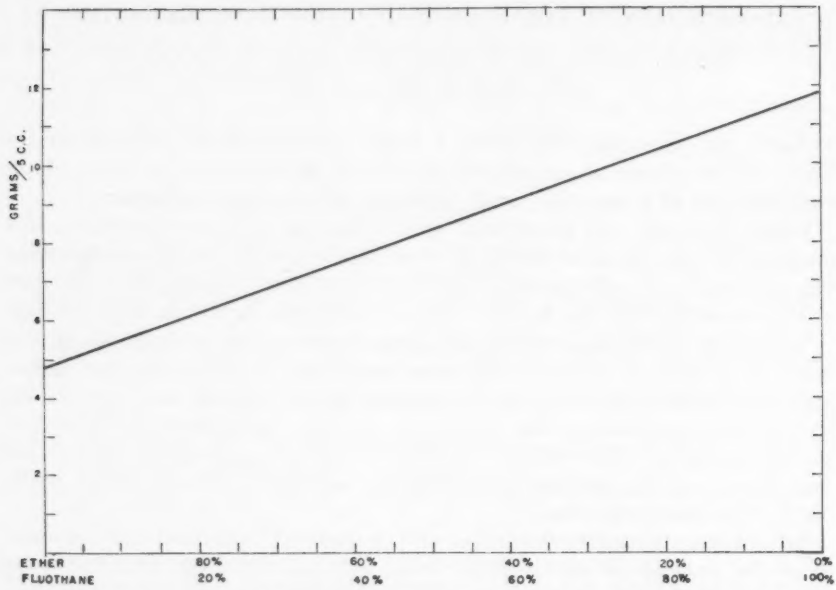

FIGURE 1. Determination of the relative proportions of ether and Fluothane from the density of a mixture measured in a 5 cc. micropycnometer.

were always in their azeotropic proportions: 31.7 per cent ether and 68.3 per cent Fluothane. It was also interesting to know the proportion of free acids produced by some decomposition of the mixture in the organism. Analyses proved that fluorine was firmly bound in the molecule when the mixture is not exposed to light. Analyses of a mixture kept in the Boyle apparatus for 48 hours did not reveal the presence of fluorine as free acid. But there was formation of very small quantities of bromine or chlorine as free acids: the proportion exhaled by the patient was not more than one part per million, a quantity already existing in the mixture inhaled by the patient.

Of the properties needed in an anaesthetic there are three which can be closely related to chemical and physical properties. With especial reference to the work of Suckling (5). These are (*a*) absence of chemical toxicity, (*b*) absence of inflammability and explosive hazards, and (*c*) anaesthetic potency.

Let us study first the problem of toxicity. One way of reducing the risks of toxicity is to work with compounds chemically inert, such as Fluothane and ether. Suckling proved that Fluothane and ether possessed a high degree of chemical stability. Analyses revealed that free acids and phosgene are not present in greater proportions than one part per ten million. Since purity of an anaesthetic must be over 99.95 per cent, the Fluothane-ether mixture with a purity over 99.99 per cent is certainly suitable. The mixture had been stabilized with thymol and kept in a brown bottle to prevent all decomposition by light. A bottle of this

mixture was left at room temperature for four months, and no decomposition was observed: the azeotropic mixture remained stable.

Several experiments were carried out to prove that the Fluothane-ether anaesthetic was not inflammable in the azeotropic proportions in clinical uses. This mixture, made of 31.7 parts of ether and 68.3 parts Fluothane, was used in the Boyle apparatus.

The Fluotec and Stephen vaporizers were opened at different degrees and then at maximum, 4.4 per cent in volume, while a current of 100 per cent oxygen was circulated, and several vapour samples were recovered by means of a special apparatus resembling an electric bulb (Fig. 2). This bulb contained an electric filament and possessed two stopcocks: one was used to remove air, and the other, to receive gas samples.

FIGURE 2. Container used to determine the gas inflammability. By means of two stopcocks it was possible to make a vacuum in the apparatus and to introduce gas samples. The wire's becoming incandescent permitted determination of the inflammability of the sample.

When electricity was turned on, it was found that vapours of this mixture were not inflammable: the bulb produced luminescence. The Fluothane, according to Seiflow (4), acts as an inert diluent when it is added to an inflammable mixture of ether and oxygen.

The azeotropic mixture was not found dangerous when mixed with oxygen in the percentage of 4.4 per cent by volume; however, it was of great interest to find the inflammability limits. For this purpose, a rubber balloon was used.

The room temperature was 25°C. and the atmospheric pressure of 741.4 mm. Hg. A volume of 4,000 cc. of oxygen measured with a rotameter was introduced into the air-free balloon and with a graduated syringe known quantities of the azeotropic mixture were added. To vaporize the azeotropic compound the balloon was heated at 52°C. in a water bath. While this gaseous mixture was at a temperature of 52°C., gas samples were taken by means of syringes. By inserting a match into the syringe, it was possible to observe if an explosion occurred. It was found that the azeotropic mixture may be mixed with oxygen in proportions of 10.7 per cent by volume without danger of explosion (Table I).

TABLE I

Az. mixture (cc.)	Gas volume at 52° C. 8741.4 mm. Hg		Az. mixture / Total mixture	Explosivity
	Az. mixture	Oxygen		
	(cc.)	(cc.)	(%)	
1.0	261.1	4362.4	5.65	−
1.2	313.3	4362.4	6.70	−
1.4	365.5	4362.4	7.73	−
1.6	417.8	4362.4	8.74	−
1.8	470.0	4362.4	9.73	−
2.0	522.2	4362.4	10.7	−
2.05	535.3	4362.4	10.9	+
2.1	548.3	4362.4	11.2	+

A mixture composed of 50 per cent Fluothane and 50 per cent ether was also vaporized in the Boyle apparatus opened at 3 per cent, not by means of a current of 100 per cent oxygen, but by one of 50 per cent oxygen and 50 per cent nitrous oxide. Samples were tested in our apparatus (Fig. 2) and it was observed there was no risk of inflammability.

After these experiments, is was of great interest to find inflammability limits of the mixture at the liquid stage. It was found that the security proportions should be 55 per cent Fluothane and not more than 45 per cent ether. To find these proportions different mixtures were prepared and tested with the flame of a Bunsen burner. It was observed that a mixture of 48 per cent ether and 52 per cent Fluothane did not inflame spontaneously, but only after 5 or 6 seconds of contact with the flame.

If such a mixture of 45 per cent ether and 55 per cent Fluothane was administered drop by drop, a period of about 2 hours was necessary to use 200 cc. and the composition did not change by more than 0.2 per cent. However, when a bottle with an opening of about 0.5 in. in diameter and containing 200 cc. of the same mixture was left opened at room temperature, a period of at least two months was necessary to evaporate all the liquid (Table II). The last cubic centimetre analysed gave the azeotropic composition: 31.7 per cent ether and 68.3 per cent Fluothane.

No matter what the proportions in which Fluothane and ether are mixed, they will always tend to volatilize in azeotropic proportions. The risks of inflammability necessarily decrease because the Fluothane concentration increases when the ether proportion decreases.

TABLE II

VAPORIZATION AT ROOM TEMPERATURE

Volume (cc.)	Time (weeks)	Ether–Fluothane (%)
200	—	49.0–51.0
180	1	46.5–53.5
155	2	41.5–55.5
105	4	41.8–58.2
85	5	39.2–60.8
50	6	37.0–63.0
28	7	33.0–67.0
8	8	31.7–68.3

The final problem was to choose compounds with adequate anaesthetic potency. Basing our researches on the work of Meyer and Hemmi (1), Mullins (2), and Robbins (3), we find that the anaesthetic potency of the azeotropic mixture was determined by the relative saturation of anaesthetic, which is given by the ratio of the partial pressure producing anaesthesia (*Pa*) to the saturated vapour pressure of the compound (*Ps*) at the temperature of the experiment. At a temperature of 24°C., the saturated vapour pressure of the azeotropic mixture was determined and it was found equal to 213.3 mm. Hg while the atmospheric pressure was 742 mm. Hg.

The partial pressure producing anaesthesia was calculated from Suckling's formula:

$$\frac{\text{concentration of the mixture} \times \text{atmospheric pressure.}}{100}$$

As the concentration used during anaesthesia was 1.5 per cent by volume, the partial pressure producing anaesthesia was equal to 11.13 mm. Hg.

In the ratio *Pa/Ps* the relative saturation was equal to 0.052 while the Boyle apparatus was giving a normal volume flow of 1.5 volumes per cent.

In clinical use considering a non-toxic and non-inflammable compound as a good anaesthetic when it is administred at a relative saturation of 0.03 to 0.08, it may be concluded that the Fluothane-ether azeotropic compound compares favourably with the best anaesthetics already known.

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LUMBAR EPIDURAL ANAESTHESIA FOR VAGINAL DELIVERY

R. A. Chaplin, M.D., and W. A. Renwick, M.D.

This is a report of the use of lumbar epidural anaesthesia for vaginal delivery at the Toronto East General Hospital. Although this method of anaesthesia has been employed in other centres, it was not utilized in this institution until the occurrence of a second death from aspiration of stomach contents. Because of the greater incidence of this catastrophe during general anaesthesia for delivery, many believe that regional anaesthesia is desirable.

Spinal anaesthesia is not acceptable to many physicians and patients. Pudendal nerve block, combined with general infiltration of the outlet, does not supply the degree of anaesthesia which we desire. Caudal anaesthesia may result in inconsistent analgesia.

Technique

Lumbar epidural anaesthesia is administered to the primipara when the cervix is fully dilated and when the head is low or when the obstetrician declares that he wishes to proceed with the delivery. In the primipara the presenting part is usually visible during a contraction. The multipara is anaesthetized when the cervix is three-fifths to fully dilated, depending upon the speed of labour. It is desirable to administer the anaesthetic at least fifteen minutes before delivery.

The many methods of identifying the epidural space and the use of different anaesthetic agents are well described in standard textbooks. The following is a brief outline of the technique which we have adopted.

Great care must be taken to maintain sterile technique. The nurse positions the patient on her side or sitting and the skin is prepared with tincture of Zephiran. No drapes are used because if the patient moves during a contraction the field may become contaminated by the shifting drapes. A skin wheal is raised with a 24-gauge needle over a suitable lumbar interspace. The epidural puncture is made using a 17- or 18-gauge Tuohy needle and the space is identified by the loss of resistance on a 20 cc. syringe partially filled with air or the anaesthetic solution. Autoclaved Xylocaine brand of lidocaine is the local anaesthetic of our choice for the procedure. It is used in a 1.5 or 2 per cent concentration with or without epinephrine. The total volume injected varies from 15 to 25 cc. This is administered in a single injection after careful aspiration to avoid subarachnoid or intravascular injection. It has not been found practical to use a test dose.

Results

As of March 28, 1958, 2,747 lumbar epidural anaesthetics for vaginal delivery have been administered in our case room. The technique varied for the first 1,747 patients, when various methods of identifying the epidural space, different sizes and types of needles, and various drugs, concentrations, and dosages were

used. Since then we have adopted the technique outlined above and the results of one thousand consecutive cases are described below.

The pain of uterine contraction is usually absent five minutes after administration and adequate anaesthesia for outlet forceps, delivery, and episiotomy is established within fifteen minutes. The premature application of and traction on forceps has necessitated supplementation with general anaesthesia.

Sensory anaesthesia usually extends to between T_7 and T_{10} (Fig. 1). The wide variation in height of anaesthesia should be noted. One patient had sensory loss to T_1. Rapid forceful injection of solution and Trendelenburg position will produce a higher level of anaesthesia.

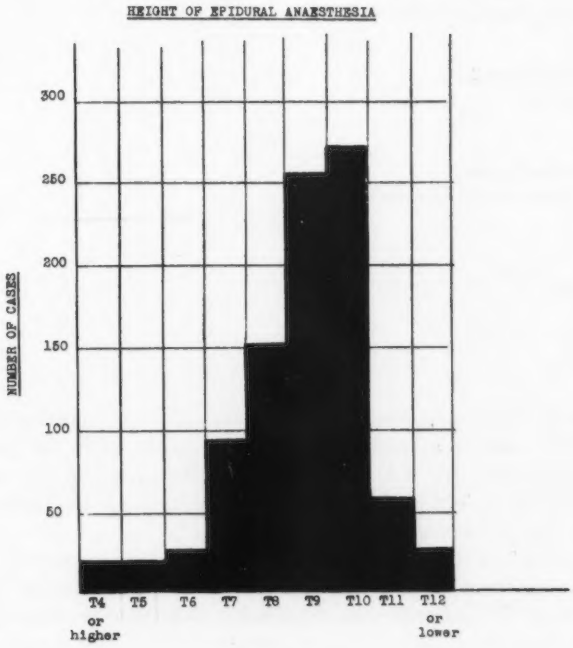

FIGURE 1

The efficiency rating of the anaesthesia is presented below:

RATING OF EPIDURAL ANAESTHESIA
(1,000 consecutive cases)

Excellent	*Good*	*Fair*	*Unsatisfactory*
772	197	17	14

This was assessed by the anaesthetist who administered the anaesthetic. The results were considered excellent when the patient felt no discomfort during delivery. A good result occurred when the patient described only a sensation of pressure or pulling. A few of these patients were given an analgesic mixture of nitrous oxide and oxygen until the head was delivered. Fair results required 50 to 75 per cent nitrous oxide with oxygen for more than the delivery of the head. Unsatisfactory results required surgical anaesthesia. This group consisted of 1.4 per cent of the total. There were 114 patients who required some inhalation anaesthetic supplement. For many of these this was necessitated by spontaneous precipitous delivery or too early application of forceps. In a few patients the epidural anaesthetic was administered too early and its effect had decreased or was gone by the time of delivery.

Complications are recorded below.

Inadvertent dural puncture	11
"Spinal" headache	4
High spinal	1
Deaths	0
Neurological complications	0
Convulsions (intravenous injection)	1
Shivering	85
Drowsiness	68
Bloody tap	11
Forceps delivery	748
Hypotension	35
Supplement	114
Not delivered	14

It is of interest that inadvertent dural puncture did not occur with two of our staff who have always administered the anaesthetic with the patient in the lateral decubitus position. It is assumed that in this position the dura is not as tense as when the patient is sitting and bending forward. Headache occurred in four patients who had inadvertent dural puncture.

In most patients a transient drop in blood pressure occurred. We did not consider this significant unless the systolic pressure was reduced to below 90 mm. Hg. There were 35 patients with significant hypotension. Among the babies belonging to this group 29 had Apgar ratings of 10; 2 had Apgar ratings of 8; 2 had Apgar ratings of 5; 1 had a rating of 3; and 1 was a stillbirth for reasons not related to the anaesthetic. The Apgar ratings of the total series are presented below.

10	9	8	7	6	5	4	3	2	1
850	56	56	12	9	10	—	5	1	1

There were 6 stillbirths, none attributable to the anaesthetic.

Included in our series were 14 sets of twins and 27 single breech deliveries.

Discussion

The graph in Figure 2 illustrates the number of vaginal deliveries under lumbar epidural anaesthesia, for a twelve-month period, compared to the number of deliveries with general anaesthesia. This shows a very receptive response to a new method of anaesthesia. One of the important reasons for this was the reaction of the patient. She was pleased to be awake and hear her baby give its first cry. She was happy to avoid the nausea, vomiting, and mental confusion

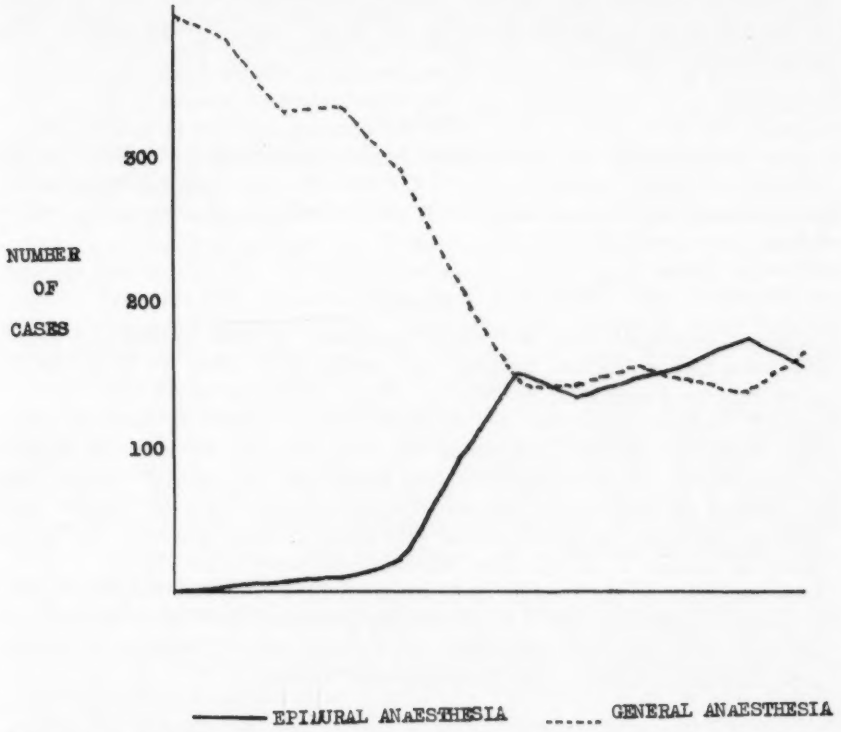

Figure 2. A comparison of the number of cases receiving general anaesthesia *vs.* epidural anaesthesia month by month (July 1, 1956, to June 30, 1957).

often associated with general anaesthesia. We have rarely encountered a patient who had previously received a general anaesthetic for labour, who did not afterwards state that an epidural was more pleasant. Many patients now request that they be given a local anaesthetic similar to that which was administered to a friend. A few have been hesitant to stay awake or receive a needle. These required some tactful explanation and reassurance that they would be put to sleep if for any reason they felt pain.

When this method of anaesthesia was first introduced into our delivery rooms, the results were not completely satisfactory because some obstetricians hesitated to use outlet forceps and anaesthetists had difficulty in knowing the optimum time for administration of the anaesthetic. Also, during this early period, the number of failures was greater because we were still acquiring the ability to identify the epidural space.

Lumbar epidural block is now our anaesthetic of choice for vaginal delivery. There are very few absolute contraindications to its use in practice. Epidural block is best avoided when there is a history of nervous system disease, a history of reaction to local anaesthetic, septic foci in the back, or bony malformation of the lumbar vertebrae.

Summary

A technique for the administration of lumbar epidural anaesthesia for vaginal delivery is described and the results of one thousand consecutive administrations are presented. Epidural anaesthesia is our anaesthetic of choice for vaginal delivery.

Résumé

En obstétrique, au cours de l'accouchement par les voies naturelles, plusieurs sont d'avis que l'anesthésie régionale est préférable à cause du risque de regurgitation et d'aspiration de vomitus. Nous pratiquons un blocage épidural lombaire environ quinze minutes avant le moment prévu de l'accouchement. Notre technique consiste à employer une seule dose de Xylocaine stérilisée à l'autoclave avec ou sans épinéphrine en employant une aiguille Tuohy. Voici les résultats de mille cas consécutifs. Quinze minutes après le blocage, nous avons pu pratiquer un accouchement sans douleur. Habituellement l'anesthésie atteint les racines T_7 et T_{10}. Dans 98.6% des cas, nous avons obtenu une anesthésie satisfaisante. Au besoin, nous avons eu recours à l'anesthésie générale. Ce genre d'anesthésie a plu à la plupart des malades. L'habileté pour pratiquer l'épidurale peut s'acquérir rapidement. Le blocage épidural lombaire est devenu notre anesthésie de choix pour l'accouchement vaginal.

TRICHLORETHYLENE ANAESTHESIA IN OBSTETRICS: A REPORT[1]

R. D. SCRAGG, M.D.[2]

TRICHLORETHYLENE was first used for maternity anaesthesia in Edmonton hospitals in 1950. Since that time over 30,000 deliveries, 19,413 of them at the Royal Alexandra Maternity Hospital, have been accomplished with the aid of this anaesthetic agent, with what we believe to be good results.

No standard premedication is used in obstetrical patients. The obstetricians make their own choice in this matter. The most common drug administered is meperidine 100 mg. with an attempt made not to administer it within two hours of delivery. A small number of patients receive meperidine 100 mg. plus hyoscine 0.4 mg. Other receive promethazine 50 mg. and meperidine 50 mg. and some get pentobarbital 90 mg. and meperidine 100 mg. All patients, with the exception of those receiving hyoscine, are given atropine 0.4 mg. approximately one-half hour before delivery.

Heidbrink gas machines are used for the administration of the trichlorethylene, nitrous oxide, oxygen mixture. The circle system of the machine is used, with the carbon dioxide absorber cut out of the circuit providing a partial rebreathing system. The soda lime is moved from the CO_2 absorber when trichlorethylene is in use to avoid any neurological complications which might result from the products of reaction between trichlorethylene and soda lime. A Heidbrink "Trimar" vaporizer is incorporated in the circle system.

If further analgesia is required during second stage labour, flow rates of N_2O 4.5 L./min. and O_2 4.5 L./min. are given to the patient by self-administration or by a nurse. The self-administration of trichlorethylene for analgesia during the early phases of labour was discontinued about four years ago. It was felt by the anaesthetic staff and the obstetrical department that the prolonged administration of trichlorethylene prior to delivery had a noticeably depressant effect on the respirations of the infant.

During the late second stage of labour when the anaesthetist takes over, the flow rates are changed to N_2O 6 L./min. and O_2 3 L./min. When the infant's head begins to distend the perineum, trichlorethylene is added to the mixture. The concentration of trichlorethylene is gradually increased and at the time that the episiotomy is performed the patient is usually unconscious. The concentration of trichlorethylene is then reduced just sufficiently to allow painless delivery and repair of the perineum. This usually means that the vaporizer is set at the second, third or fourth notch, with a total gas flow still about 9 L./min. The deepest level of anaesthesia obtained corresponds to plane 1 of stage 3.

Of the infants born under this technique 75 per cent require no care except for drainage of mucus by posture and suction. These infants cry immediately after delivery. Of the remainder 20 per cent are given oxygen to overcome cyanosis. The remaining 5 per cent may require some mechanical assistance

[1]Presented at a meeting of the Western Divisions, Canadian Anaesthetists' Society, Calgary, Alta., March 13, 1958.

[2]Royal Alexandra Hospital, Edmonton, Alberta.

to attain normal respiratory activity and colour. The 25 per cent with respiratory difficulties includes those cases where labour was difficult and also the difficult forceps deliveries.

Blood loss during the third stage of labour is minimal in the opinion of the obstetrical staff and no greater than with other techniques.

Ergotrate 0.25 mg. is usually administered intravenously when the anterior shoulder descends under the public arch.

Following the termination of the anaesthetic the majority of patients are awake and talking within five minutes.

All cases, including difficult mid-forceps rotations and extractions, are managed by this technique although we usually prefer low spinal anaesthesia for the latter when we are forewarned of trouble. Breech deliveries are also managed with trichlorethylene and nitrous oxide but with very low concentrations of the former to retain the patient's co-operation during the delivery. We prefer to administer pudendal blocks to these patients. Patients requiring relaxation of the uterus during delivery are given ether. Caesarean sections are managed with spinal or general anaesthesia.

Complications arise with trichlorethylene just as they do with any anaesthetic agent. Nausea and vomiting are frequently seen, especially in patients with full stomachs, and give some cause for concern. The routine use of ergotrate may have something to do with this. Fortunately, the patients are nearly always awake when this occurs and, if not, they are in possession of their protective reflexes. In our series we have encountered only one patient with an acid-aspiration syndrome and she recovered without special therapy. There are no cases on record in the Royal Alexandra Hospital of pneumonia following aspiration of stomach contents after trichlorethylene anaesthesia. Three cases of aspiration occurring in one of the other hospitals, each resulting in severe pneumonitis. Each of these patients recovered.

Partial respiratory obstruction is often a problem with certain patients. These patients hold their mouths tightly shut during anaesthesia, making insertion of an oral airway difficult, and many of them have a degree of nasopharyngeal obstruction. The passage of a well-lubricated nasopharyngeal tube resolves this problem. Laryngeal spasm may occasionally occur to some degree when the perineum is put on the stretch as the foetal head is crowning. It disappears after the birth of the head.

The expiratory phase of respiration may be very noisy owing to the blowing of air through the closed lips.

Tachypnoea may occur from overdose of trichlorethylene. Reduction of the concentration overcomes this complication.

Bradycardia was a frequent finding before the routine use of atropine, with rates as low as fifty per minute occasionally seen. It is seldom encountered now. Occasional irregularities in rhythm are noted, especially if the concentration of trichlorethylene is too high.

Since adrenalin may produce ventricular fibrillation during trichlorethylene anaesthesia it is forbidden to keep mixtures of adrenalin and local anaesthetic agents in the maternity hospital. This prevents accidental administration which might have tragic results.

Agitated, nervous women are sometimes quite resistant to trichlorethylene. I can recall one patient who would not go to sleep in spite of a high concentration of the agent. Intravenous injection of 50 mg. of Demerol at this point transformed it into a very easy anaesthetic and the baby was born shortly thereafter. Usually, a little gentle persuasion and some patience overcome this problem.

Convulsions during anaesthesia have been rare. This complication occurred in four patients. The cause was not determined. One patient convulsed twenty minutes after awakening and no cause could be determined.

The total number of deliveries at the Royal Alexandra Maternity Hospital from January 1, 1954, to January 1, 1958, was 19,645. Of these 90 per cent were done under trichlorethylene anaesthesia. Forceps were used in 33 per cent of the deliveries.

In this series two of the mothers died, one from exsanguination following rupture of the uterus from internal version done under ether and the other from a pulmonary embolus three days after a Caesarean section done under spinal anaesthesia. There have been 448 cases of maternal morbidity, none of which could be attributed to trichlorethylene.

The perinatal death rate averaged over the 4-year period is 26.01/1,000 (provincial, 23.9/1,000) births which includes all of the prematures, whether they were considered viable or not. It also includes the congenitally deformed stillborn infants.

Perinatal Death Rate

1954	$\frac{126}{4,511}$	= 27.95 per 1,000 births
1955	$\frac{125}{4,795}$	= 26.06 per 1,000 births
1956	$\frac{126}{5,112}$	= 24.64 per 1,000 births
1957	$\frac{134}{5,227}$	= 25.63 per 1,000 births
Total	$\frac{511}{19,645}$	= 26.01 per 1,000 births

The neonatal mortality rate[3] is 14.37/1,000 live births.

Neonatal Mortality Rate

1954	$\frac{75}{4,460}$	= 16.81/1,000 live births
1955	$\frac{66}{4,736}$	= 13.93/1,000 live births
1956	$\frac{65}{5,051}$	= 12.86/1,000 live births
1957	$\frac{73}{5,166}$	= 14.13/1,000 live births
Total	$\frac{279}{19,413}$	= 14.37/1,000 live births

[3]This figure includes all live born babies, prematures, etc., who subsequently died in hospital.

The number of premature infants (under five pounds) was 1,211 or 6.06 per cent. The perinatal death rate for premature infants is 289 per 1,000 births (provincial 233). The neonatal mortality rate in premature infants is 187.7 per 1,000 live births.

We recognize the fact that trichlorethylene leaves a lot to be desired as far as a perfect anaesthetic agent is concerned. However, the low maternal death rate, the paucity of serious complications, and the favourable infant death rate in this series leads us to believe that trichlorethylene is a safe anaesthetic agent for obstetrical use.

This method of maternity anaesthesia is used in the four Edmonton hospitals. There have been no maternal deaths attributable to trichlorethylene.

The University of Alberta Hospital has had over 10,000 deliveries since 1950 and their neonatal death rate was 2.9 per cent. The perinatal death rate in this series was 1.6 per cent.

The Edmonton General Hospital has a series of 5,000 deliveries from July, 1955, to December, 1957. Their neonatal death rate was 3.03 per cent and the perinatal death rate 1.8 per cent.

Summary

A series of over 30,000 maternity cases, in which trichlorethylene was used as the main anaesthetic agent is presented.

There was no maternal death attributable to the anaesthetic technique. Of these cases 19,645 were done in the Royal Alexandra Hospital in Edmonton. The perinatal death rate was 2.6 per cent. The neonatal death rate was 1.4 per cent. At the University of Alberta Hospital, where 10,000 cases were done, the neonatal death rate was 2.9 per cent. The perinatal death rate was 1.6 per cent. In the Edmonton General Hospital, where 5,000 cases were done, the neonatal death rate in this series was 1.8 per cent and the perinatal death rate was 3.03 per cent. These death rates are uncorrected for such things as congenital deformities.

Résumé

Nous avons présenté une étude de 30,000 cas de maternité où nous avons employé le trichlorethylene comme agent anesthésique principal.

Aucune mort maternelle n'a été attribuable à la technique anesthésique.

A l'hôpital Royal Alexandra à Edmonton 19,645 cas ont été faits. Le taux de mortalité périnatale a été de 2.6%. Le taux de mortalité néonatale a été de 1.4%. A l'hôpital de l'Université d'Alberta 10,000 cas ont été faits. Le taux de mortalité néonatale a été de 2.9%. Le taux de mortalité périnatale a été de 1.6%. A l'hôpital Edmonton General 5,000 cas ont été faits. Dans ce groupe, le taux de mortalité néonatale a été de 1.8% et le taux de mortalité périnatale a été de 3.03%. Ces taux de mortalité ne sont précis à cause de facteurs, tels que les déformités congénitales.

THE MANAGEMENT OF A POST-POLIOLYTIC PATIENT FOR MAJOR ABDOMINAL SURGERY

M. MINUCK, M.D. and R. S. LAMBIE, D.A. (ENG.)[1]

A SEVERELY HANDICAPPED PATIENT was recently presented to us for a cholecystectomy. His disability posed an interesting problem in anaesthetic management, for he suffered from a complete residual paralysis of all four limbs, the abdominal wall, and the costal muscles as a result of acute poliomyelitis which he had contracted in August, 1953.

After perusal of the literature we were unable to find any details of pre-operative, operative, and post-surgical management for this type of case. For this reason we feel that this presentation may be of interest.[2]

CASE HISTORY

The patient, a 52-year-old white male, was admitted to St. Boniface Hospital on November 11, 1957, with a diagnosis of cholelithiasis and chronic cholecystitis. He gave a typical history extending over two years, with increasing frequency of attacks of pain in the preceding few months.

His previous surgical history was as follows: an appendectomy in 1945, a gastrectomy in 1950, and a mastoid revision in April, 1953. All results were satisfactory.

In August, 1953, he developed acute anterior poliomyelitis which required management in a respirator for eight and a half months. Paralysis was permanent and complete, extending from D_4 downwards. He was eventually discharged in 1954.

Physical examination of the chest wall showed wasting of the intercostal muscles. His diaphragmatic movement was good. The lung fields were clear. His vital capacity was 2,235 ml., or 58 per cent of his normal predicted value. His timed vital capacity was normal, indicating that his pulmonary insufficiency was due to muscular atony and not to bronchospasm. His maximum breathing capacity was 47.6 L./min. An X-ray of his chest showed left ventricular prominence, and elongation and broadening of the aorta. Linear atelectasis was noted at the left base, and there was evidence to suggest bronchiectasis in the left lower lung. Emphysematous bullae were noted in both apical regions, more marked in the right. Except for an elevated sedimentation rate (76, Westergren), his blood picture was normal.

ANAESTHETIC MANAGEMENT

For sedation, the night prior to operation, he was given Tuinal® 100 mg. orally. One hour before the operation he received promethazine 50 mg., and atropine sulphate 0.4 mg. intramuscularly.

One litre of 5 per cent glucose in water was started intravenously. The patient was then induced with 150 mg. of thiopental sodium, followed by 15 mg. of succinylcholine. At the same time 100 per cent oxygen was delivered by mask. When paralysis was complete, the larynx was sprayed with 2 ml. of 2 per cent

[1]St. Boniface Anaesthetic Clinic, Winnipeg, Man.

[2]The authors wish to thank Dr. S. S. Peikoff for permission to discuss his patient in this paper.

Pontocaine Hydrochloride®, and intubated with a number 10 cuffed Magill endotracheal tube. He was maintained on a semi-closed system of nitrous oxide and oxygen, in a 4:2 litre mixture, and intermittent doses of thiopental. Small increments of succinylcholine were given to allow controlled respiration, using the Jefferson respirator. The latter was set to exert a positive pressure of 15cm. H_2O and a negative pressure of 5 cm. H_2O at a rate of 20 per minute.

The small intermittent doses of thiopental were given to supplement the nitrous oxide in order to maintain the stage of analgesia. The eyelash reflex was present throughout the operation which lasted one hour and fifteen minutes.

A total of 400 mg. of a 2½ per cent solution of thiopental and 60 mg. of succinylcholine were used.

His pulse and blood pressure were recorded every five minutes. At the beginning of the operation the systolic pressure was 120 mm. Hg and his pulse 100 per minute. Upon completion it was 110 mm. Hg and 85 per minute. There were no fluctuations recorded during the procedure.

After the peritoneum was closed, one polyethylene tube was placed along the rectus sheath, and two others at each corner of the incision. The free ends were taped clear of the incision and dressing, and through each 5 ml. of a 2 per cent solution of Procaine were injected.

A 3 ml. mixture, containing 100 mg. of piperidine plus 1.0 mg. of levallorphan, was prepared; 1 ml. of this was given intramuscularly 25 minutes prior to the end of the operation.

After a careful supralaryngeal toilet, the endotracheal tube was removed. The patient was conscious but drowsy, and complained of moderate pain. A further 0.3 ml. of the piperidine-levallorphan mixture was given intravenously.

Over a period of 6 minutes 150 mg. of B-methyl B-ethylglutamide (Megimide) were given intravenously. Very shortly after the administration of this drug the patient was completely awake, exhibiting no drowsiness, and showed return of his usual diaphragmatic excursions. The patient was transferred to the post-anaesthetic room in excellent condition.

Postoperative Management

For the control of pain the patient received the following drugs: (*a*) 5 ml. of 2 per cent Procaine were injected into each of the polyethylene tubes—this was required approximately every 4 hours; (*b*) Piperidine 50 mg. plus 0.5 mg. of levallorphan—this was needed 15 times throughout the 16 days of postoperative period.

To combat pulmonary complications, aminophylline suppositories, bronchodilators, and physiotherapy were continued throughout the postoperative period.

Arterial blood pH and CO_2 content and CO_2 tensions were determined on the first and second postoperative days and found to be within normal limits. On the fourteenth day the vital capacity and maximum breathing capacity were somewhat reduced, compared with the preoperative record.

The patient was discharged sixteen days after his operation.

Discussion

The marginally existing patient, such as we have here, is intolerant of any further discomfort or pain, superimposed upon his present disability. He is naturally much concerned about the outcome of his proposed major surgery. He is psychologically and physically an anaesthetic challenge (1).

The problem was to decide what anaesthetic technique would allow surgical intervention with the minimum of physiological imbalance, particularly of the respiratory system, and allow pain-free normal respiratory function in the post-operative period.

Crasilneck has suggested hypnosis or hypno-analgesia be used in such cases (2). Although we feel that hypnosis has a definite role in anaesthesia, we did not think that this particular patient was suitable for hypnosis nor was our experience in this method sufficient.

Explanation and assurance did not remove a deep-seated anxiety concerning his operation. For this reason, plus the usual contraindications for high spinal or epidural analgesia, these regional techniques were thought to be unsuitable (3). Thiopental was used for induction in order to avoid excitement and psychological trauma and further to ensure a smooth induction. Nitrous oxide was the obvious choice of inhalational agent for it is non-explosive and non-irritative, and can be used in high flows with the Jefferson respirator. Our aim was not to establish anaesthesia but to remain in plane 3 of the stage of analgesia (4). Ideally, verbal contact should be upheld in order to be certain that the patient is in analgesia. In this case slight movements of the facial muscles were present, as well as a somewhat reduced eyelash reflex. Through previous experience these signs were presumptive evidence of analgesia. Several workers have demonstrated the value of this anaesthetic technique, especially for the poor risk and geriatric case (5, 6). It has been found that there is minimal depression of the cardiovascular and central nervous systems, with marked reduction in reflex activity (7).

The small dose of succinylcholine was sufficient to allow smooth endotracheal intubation, and controlled respirations with the Jefferson respirator. As the total dose was 60 mg., there was little likelihood of immediate postoperative respiratory depression.

Some may object to the use of Megimide in the attempt to reverse the effect of thiopental, and it is admitted that there is a great deal of controversy concerning the exact mechanism of Megimide. Recent studies have suggested its use to "reverse" thiopental anaesthesia, and have demonstrated its clinical advantage (8, 9). One of us (R. S. L.) used this drug to reverse buthalitone sodium anaesthesia on out-patients almost daily for one year with excellent results. Doses from 500 mg. to 1,000 mg. of buthalitone were counteracted by doses of 50 mg. to 200 mg. of Megimide. This drug is probably not a specific barbituate antagonist, but in some way it performs its counteraction by central stimulation. This action is unlike that of analeptics, which raise the blood pressure and induce hyperpyrexia. There can be no doubt concerning its dramatic effect upon reflex activity and respiration following barbiturate anaesthesia, which was clearly demonstrated with this patient.

After the operation the patient immediately gave verbal indication of our next problem: the control of his postoperative pain. It was realized that if the usual dose of narcotic was used the tidal volume would be seriously reduced; on the other hand, if it was withheld, the vital capacity would still be reduced through splinting of the diaphragm.

Eckenhoff has pointed out that it is important in the patient with normal respiratory function to avoid the development of respiratory acidosis (10). In this case it was vital to avoid tidal volume reduction either by under- or by oversedation.

Several opiate antagonists have recently been synthesized, and we have had the opportunity to use levallorphan tartrate (Lorfan®) in conjunction with narcotic-supplemented anaesthesia, and also in the postoperative period. Although the mode of action of these antagonists has not been clarified, reports show that they do reverse the respiratory depression (11, 12). These drugs, we feel, are a great step forward in the endeavour to allow pain to be relieved without the concomitant respiratory depression.

For the first two days after the operation local wound analgesia was successfully achieved by the already described polyethylene tube irrigation: 33.3 mg. of piperidine combined with 0.33 mg. of levallorphan were administered 5 times in the first 48 hours after the operation. We felt that this small amount of narcotic given for pain relief indicated the efficacy of the wound analgesia.

The authors realized that the major problem would arise in the postoperative period when depressive analgesia would be necessary to control severe pain. A tank respirator was kept on the ward should it become necessary, but at no time did the patient's condition warrant its use.

Summary

The anaesthetic management of a severely handicapped post-poliomyelitic patient for major surgery has been described. The method is described in detail, with comment on the operative and postoperative management.

Emphasis has been placed on a rapid return to consciousness following surgery, and to pain-relief from the abdominal wound without respiratory depression. This was accomplished by two methods: (*a*) irrigation of the wound with local anaesthetic solution as required, (*b*) the use of a Pethidine-Lorfan mixture.[3]

Résumé

Nous avons essayé de présenter la technique anesthésique et le ménagement post-opératoire d'un patient atteint d'une sevère insuffisance pulmonaire et dont l'état nécessitait une cholécystectomie.

L'insuffisance pulmonaire de ce patient est le résultat de la poliomyélite subie en 1953. L'examen médical et les études sur la fonction pulmonaire ont révélé la diminution de sa capacité vitale et respiratoire.

[3]This mixture is now available in a ready-mixed ampoule, Pethilorfan®.

L'anesthésie se composait de Pentothal, de nitride oxide, d'oxygène et de meperidine. L'effect dépressant de Pentothal a été reversé par Megimide, l'indésirable action de meperidine sur la respiration a été reversé par levallorphan. Succinyl choline HCl a été employé pour obtenir la relaxation musculaire.

Les soins post-opératifs qui impliquaient le soulagement de la douleur sans toutefois réduire davantage le capacité vitale du patient ont été administrés de la façon suivante: (*a*) les cathéters polyethelene, qui étaient placés dans l'incision lorsque celle-ci fut close, ont été irrigés avec 2% de Procaine; (*b*) 50 mg. de meperidine plus 0.5 mg. de levallorphan ont été administrés pour le douleur. Finalement une vigoureuse physiothérapie a été administré.

La guérison s'est produite sans imprévus et l'étude post-opérative de la fonction pulmonaire n'a indiqué aucune signifiante réduction comparée à celle faite d'avant l'opération.

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FEMORAL NERVE INJURY FROM ABDOMINAL RETRACTORS

F. G. Ruston, M.D., and V. L. Politi, M.D.[1]

It is obvious from the title that this paper should be directed to a surgical group instead of to the "Sleeping Partners," as anaesthetists have been so aptly labelled by Punch (1). However, the etiology of nerve lesions is important to the anaesthetist, especially if spinal, epidural, or some other form of conduction anaesthesia has been used. The following examples of femoral nerve injury by abdominal retractors are presented to illustrate this point.

Case Presentations

Case 1

A female patient, age 28 years, was admitted to the Hamilton General Hospital on August 15, 1954, for an abdominal hysterectomy to terminate a three and one-half months' pregnancy. The medical consultants advised this as the patient was experiencing increasing respiratory distress and morning sickness. These symptoms had started three weeks prior to admission and were characterized by fatigue, shortness of breath, non-productive cough, and wheezing in her chest. She had not been troubled like this in her previous pregnancies.

The past history is important, for she had pleurisy with effusion in 1942 and was hospitalized in a western sanatorium. In 1944 she had an eight-rib thoracoplasty to collapse a tuberculous cavity. In 1945 she was discharged from the sanatorium and, following this, she had three viable pregnancies. She was hospitalized again for pneumonia in 1950. The last pregnancy was complicated by pyelitis, and finally in 1953, she had a nervous breakdown.

On examination the evening before operation it was noted that the patient was quite apprehensive. The moral issue of terminating the pregnancy was troubling her. She was a thin woman, showing some dyspnoea without cyanosis, and coughing occasionally. There was a residual deformity of the left chest from the thoracoplasty. Râles and rhonchi were present throughout the whole chest. The blood pressure and heart were normal. Laboratory reports were negative except for the haemoglobin, which was 62 per cent.

The patient wished to have a spinal anaesthetic since as a result of her previous anaesthetics she had developed a fear of losing consciousness.

On August 19, 1954, the patient received a preoperative sedation of Seconal® gr.iss, morphine sulphate gr. 1/6, and hyoscine gr. 1/150. She was normally sedated, co-operative, and reminded me of my promise not to put her to sleep. She received a spinal anaesthetic, the tap being made between lumbar interspaces 3 and 4, and 12 mg. of Pontocaine® with 10 per cent dextrose were injected. Anaesthesia and relaxation were good and no supplement was required, although meperidine 25 mg. was given intravenously to further sedate the patient, as she was talkative. She was content and did not have any discomfort during the spinal tap or the operation.

The surgical approach was through a low transverse incision in which the recti muscles were not divided. A Mann self-retaining retractor[2] was used with the two fixed lateral blades in the usual position for pelvic laparotomies. The hysterectomy was performed without difficulty and without excessive bleeding, although a transfusion of

[1]Hamilton General Hospital, Hamilton, Ontario.

[2]The Mann self-retaining retractor was designed by Dr. John Mann, Toronto, and is manufactured by Imperial Surgical Company, Toronto, Ontario.

500 cc. of matched blood was administered during the operation. There was no bleeding present at the time of wound closure.

An additional 500 cc. of blood was given to the patient postoperatively. She complained of abdominal pain which meperidine 100 mg. would not control. However, Dromoran® 2 mg. proved effective. Two days later, the patient complained of numbness and weakness in her left leg. This was more marked on the third postoperative day and was accompanied by abdominal distention, pallor, and dyspnoea. A Levine tube was inserted into the stomach and continuous gastric suction was started. The haemoglobin fell to 48 per cent and she was transfused again. The surgeon called a neurosurgeon in consultation. Here are excerpts from his report. "*The day before I saw her, she found that she could not extend her left leg at the knee. At the same time she was distended and had an ileus. It is my opinion that a certain amount of haemorrhage had occurred in the retroperitoneal space. In my opinion the weakness of the leg is due to the blood clot causing pressure on the lumbar roots going to make up the femoral nerve. It is quite easy to imagine a blood clot pressing on these roots beneath the peritoneum over the psoas muscle. On this examination of August 22nd, there was a patch of analgesia in the lower L3 dermatome and hyperaesthesia on part of L4 dermatome. She could not extend her leg at the knee, although the quadriceps did contract slightly on the lateral side. The left knee jerk was almost gone. On August 25th, the leg could be extended weakly and the sensory loss was less. In my opinion, this incident was merely a complication of the operation which will be corrected when the blood clot has been absorbed. I do not see how the spinal anaesthesia could be implicated. I think she will make a good recovery in a few weeks' time.*"

There was a gradual decrease in numbness and an increase in quadraceps power following a course of physiotherapy. She had completely recovered from neurological symptoms at the end of five months.

Case 2

A female patient, age 33 years, had been observed for some time because she had symptoms of pelvic disease which were clouded by a marked emotional background. After an additional gynaecological consultation, it was decided to hospitalize her and do a diagnostic curettage and a pelvic laparotomy. The patient was subjected to these procedures on October 29, 1955, under combined spinal and thiopentone anaesthesia. The spinal tap was difficult and, on the first attempt, the patient experienced a stabbing pain down her right leg. The spinal needle was reinserted and good anaesthesia resulted from the injection of Pontocaine® and dextrose. Following the curettage, a left paramedian incision was made and a Mann retractor was put in place in the usual manner. The patient was placed in a very steep Trendelenburg position in which the thighs were hyperextended and a presacral neurectomy, bilateral ovarian neurectomy, and removal of a papilloma from the right ovary were done. The abdomen was closed after numerous plaques of endometriosis had been cauterized. As soon as the patient had recovered from her anaesthetic, she complained of numbness and weakness in her left leg. This persisted and she was examined by an internist ten days later on November 9. Here is a quotation from his report. "*Noticed inability to move left leg from the knee down after regaining consciousness. The leg felt like a log and numb. Numbness was on inner side of thigh and calf and is gradually disappearing and she is aware of tingling with the return of power. On examination, there is a reduced touch sensation—she can feel but cannot differentiate over the inner side of thigh and calf. Pain sensation-sharpness is reduced over this area. Vibration sense is O.K. The right knee and both ankle jerks are very active, but the left knee jerk is markedly reduced. Extension of knee and flexion of ankle is greatly reduced. Muscles tighten very markedly but movement doesn't occur against the slightest resistence. I suggest some nerve damage but a large functional overlay is present.*"

A note made on November 18, nearly three weeks after operation, stated that "*Flexion of the ankle is practically normal but extension of the knee is still poor. Can differentiate*

sensation and reflexes are normal. She gets up by herself but tends to drag her left foot after the first few steps."

She was examined by a surgical consultant on November 20. *"There is a diminished sensation on the medial side of the thigh and calf and somewhat reduced left knee jerk—some weakness of left quadraceps and adductors. I believe that this represents stretching or contusion of the femoral-obturator nerve and this will almost certainly recover. Suggest no other treatment than her present physiotherapy and encouragement."*

She was discharged to her home on December 10, having been in hospital ten weeks. Progress notes from her record after she had left the hospital are as follows. *"Muscle power in left leg considerably improved but there is still some weakness."* There was no further mention of the leg when she was examined on April 13, 1956. She was re-examined on several occasions regarding endometriosis, the last time being August 19, 1957, and there was no further reference to her leg.

Case 3

A woman, age 38 years, was admitted on March 13, 1955, with the complaint of dysmenorrhoea of increasing severity during the past year, especially at the beginning of the menstrual period, low back pain, and, more recently, of stress incontinence. Physical examination was essentially negative, except for the findings of vaginal examination. Blood pressure was 130/80, haemoglobin, 60 per cent, white blood count, 9,600, and W. R., negative.

On March 15 she came to surgery and, after an examination under anaesthesia, it was decided that a hysterectomy should be done by the abdominal approach. Anaesthesia was induced with 300 mg. of thiopentone and maintained with cyclopropane and oxygen in closed system. The necessary relaxation was obtained with Tubarine® 9 mg. The length of anaesthesia was 1 hour and 50 minutes. She received 500 cc. of 5 per cent dextrose in distilled water during and following the operation. Pulse and blood pressure remained steady in the region of 72 and 110 mm. of mercury, respectively. She arrived in the recovery room in good condition and returned to the ward in a short time.

The gynaecologist described the approach as *"a transverse muscle cutting incision."* The Mann abdominal retractor was inserted in the conventional position. The operation was a total hysterectomy with left salpingo-oopherectomy and appendectomy. At 3.30 P.M. she was transfused with 500 cc. of cross-matched blood along with additional fluids. She developed abdominal distention and this was treated with Prostigmine and fluids.

On March 18, two days after operation, she complained about her legs being painful. The following day she had numbness in her right leg and diminished feeling in her left. One week later her leg collapsed when she got out of bed and she fell on the floor. On March 26 she complained of increasing discomfort in her legs. For the next four weeks she continued to have difficulty in moving about the ward. She had to be assisted in walking. She walked as though on tip-toes. During this time there were numerous reports of falling while walking.

She was examined by one of the staff surgeons, but no record was made of his findings except that she should be placed on anticoagulants and, later, physiotherapy. She was seen by the anaesthetist the day following the operation and was found to have recovered from the anaesthesia. She was not seen again until the gynaecologist reported that the anaesthetist had caused a *"paraplegia with your spinal anaesthetic."* She was examined superficially regarding the muscular weakness in her legs. Her chief difficulty was that she could not keep her legs straight or raise them from the bed. Extension of the knees could not be carried out well though she could dig her heels strongly into the mattress.

She remained on physiotherapy for some time and was finally discharged from hospital on May 7, 1955, after a stay of six weeks. She continued to have difficulty in walking for several months but has since fully recovered the use of her legs.

A discussion of the case with the individuals concerned gave a diagnosis of bilateral femoral nerve paralysis due to trauma. It is regretted that a full neurological examination of the lower extremities had not been recorded on the consultation report.

Discussion

A review of the literature on femoral nerve injuries over the past five years is surprisingly meager. However, there is one recent article, "Nerve Injuries Incident to Anesthesia and Operation," by Nicholson and Eversole (2), which, while it covers other peripheral nerves, only refers to the femoral or anterior crural nerves in a table, "Incidence of Peripheral Nerve Injuries," which lists figures from a text dated 1933 (3). In this table there were 19 femoral nerve injuries out of a total of 729 peripheral nerve injuries from all forms of trauma. In a modern text, *Neurosurgery in General Practice* (4), even injuries of the peripheral nerves of the lower extremities are covered by one sentence, "Lesions of these nerves are relatively rare in civil practice and they will not, therefore, be discussed here." So it may be assumed that reports of femoral nerve injuries are comparatively rare in medical literature. Perhaps the reason that injury is not more frequent is that care is used by the surgeons because of the proximity of the iliac vessels. However, the femoral nerve is vulnerable to direct pressure and stretching because of the exposed relationship to the psoas muscle in the portion proximal to the femoral canal.

Figure 1 shows the schematic distribution of the femoral nerve. Figure 2 plots the dermatomes supplied by this nerve. It is evident that these figures are applicable to the pattern of involvement of the three cases mentioned above.

There was a rather reasonable, yet quite understandable inference drawn from the complications in these patients. Each was originally thought to have been a complication of spinal anaesthesia. The first was initially believed by the surgeon to be a cauda equina syndrome. The anaesthetist also had some misgivings until the etiology became evident. It seemed that the picture was one of direct pressure on, or stretching of, the femoral nerve. An extraperitoneal haemorrhage also developed after the removal of the retractor, because there was no evidence of bleeding or haematoma prior to closure of the incision. Examination of the Mann retractor (Fig. 3) shows that the two lateral fixed blades have sharp edges (Fig. 4) which would impinge on the femoral nerves in the low transverse incision. An example of this is seen in Figure 5. This was discussed with the gynaecologist and the neurosurgeon and they agreed that this explanation was reasonable.

In case 2 the patient believed that her numbness and weakness resulted from the initial spinal tap. She had complained of a shooting pain down her right leg, whereas the left leg was the affected one. A paramedian incision was made and a steep Trendelenburg position with hyperextension of the thighs was used. Figure 6 shows the lateral blades resting on the psoas muscle in the area of the femoral nerve. It must be pointed out that the head of the table was only slightly tilted for this photograph.

Case 3 developed bilateral femoral nerve involvement. The surgeon greeted the anaesthetist with "Well, it's happened again, paraplegia with your spinal anaesthetic." This time the answer was "Why, I gave her a general anaesthetic"!

Fortunately, all these patients have fully recovered. Moreover, there is no doubt in the minds of the surgeons and the anaesthetists that the cause of

the femoral nerve trauma was pressure exerted by the Mann retractor when it was inserted in the usual manner for pelvic laparotomies. Doctor Mann stated, at an informal discussion, that the retractor was not designed for the low transverse or Pfannenstiel's incision. Doctor R. T. Weaver (5) emphasizes that the

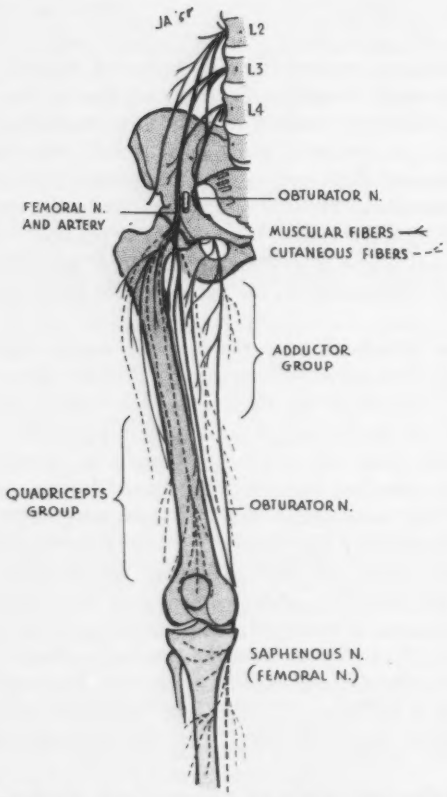

FIGURE 1. Schematic distribution of femoral nerve.

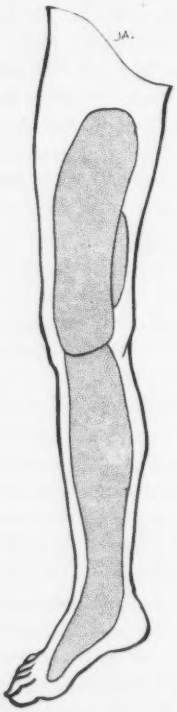

FIGURE 2. Lumbar 3 and 4 dermatomes supplied via femoral nerve.

Mann retractor should be used in the reversed position for pelvic laparotomies in either the vertical or the low transverse incisions so that the lateral blades do not press on the femoral nerves or the iliac vessels. Moreover, he releases the ratchet momentarily during the operation so that no unduly prolonged pressure will be placed on the incision. In his opinion, the Mann retractor offers maximal harmless exposure if these precautions are practised. Figure 7 shows the safe position for the low transverse incision; the point of the forceps is over the femoral nerve and the retractor blade is lateral to this. Figure 8 demonstrates the safe position for the vertical approach.

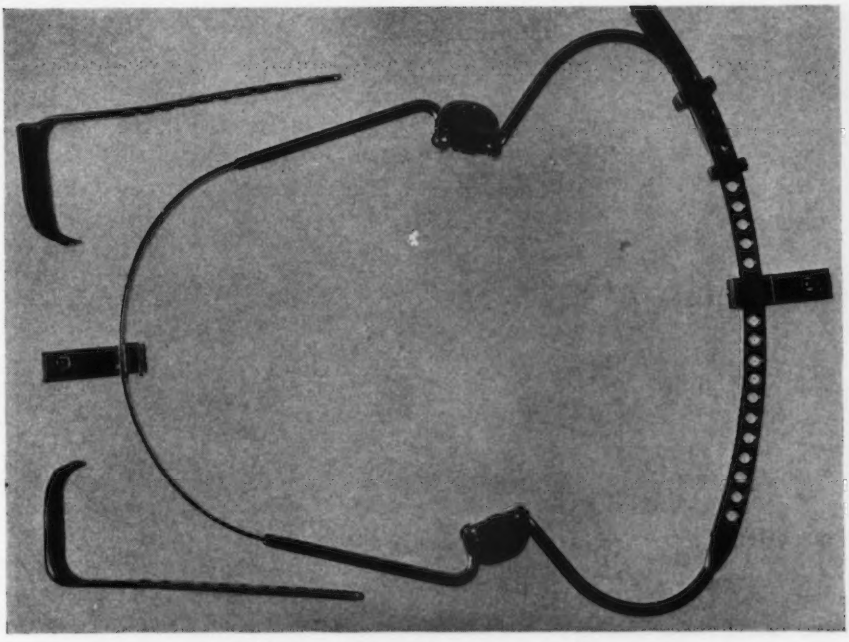

FIGURE 3. Mann self-retaining retractor.

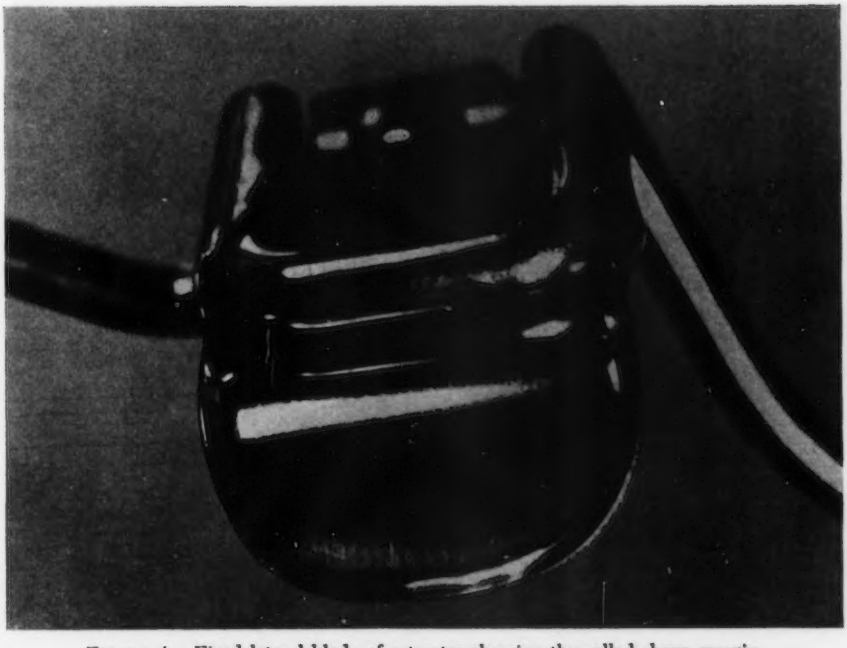

FIGURE 4. Fixed lateral blade of retractor showing the rolled sharp margin.

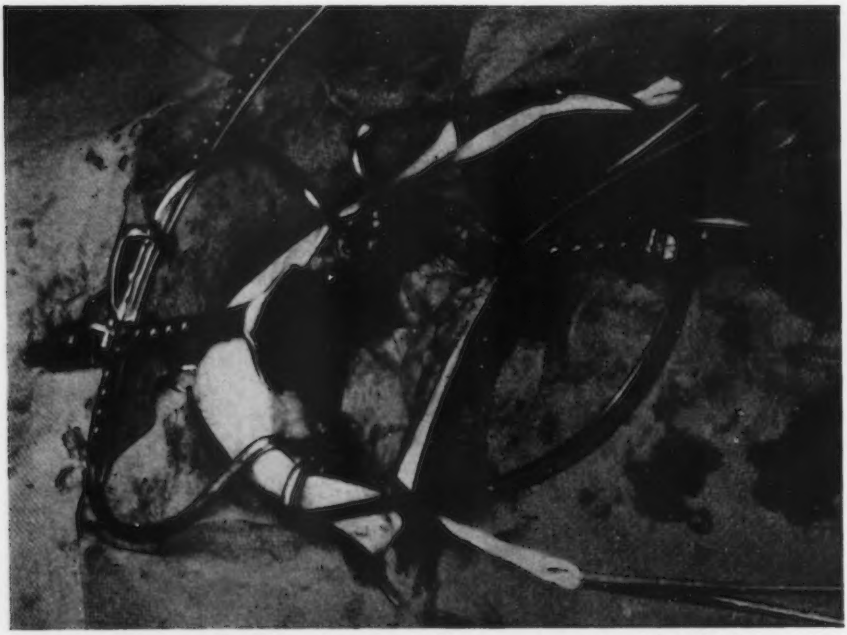

FIGURE 5. Conventional position of retractor in low transverse incisions—large blade on pubes.

FIGURE 6. Mann retractor in conventional position for vertical incision.

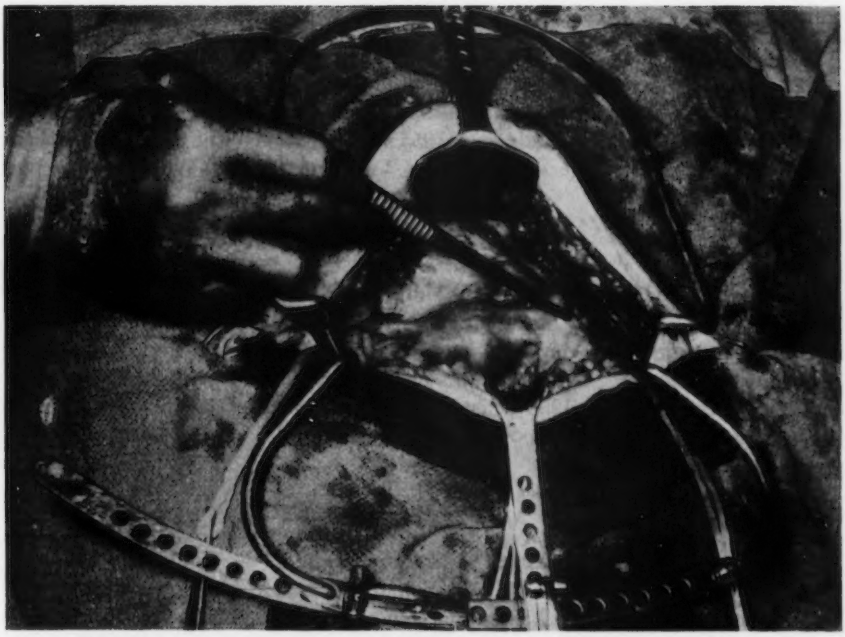

FIGURE 7. Retractor reversed—large blade on pubes—low transverse incision.

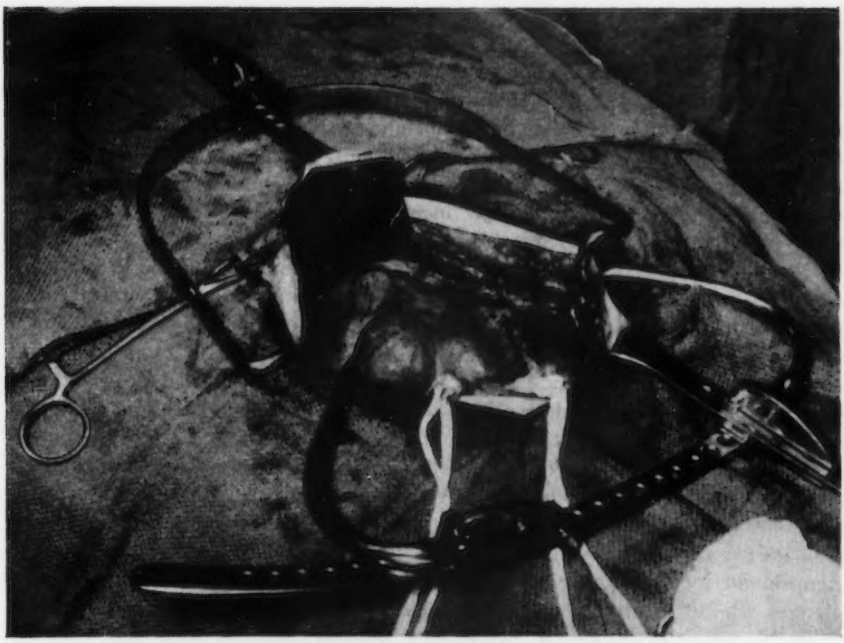

FIGURE 8. Safe position of retractor for vertical incision.

It is significant that one of the gynaecologists has abandoned the use of this retractor because of the neurological complication which was sustained by his patient. It is also interesting that there has been no recurrence of femoral nerve trauma in the Hamilton hospitals since 1955 when the danger of the usual position was recognized and the modifications suggested by Doctor Weaver were put into practice.

Summary

Three patients who had pelvic laparotomies have been presented. These showed transient femoral nerve involvement following the use of the Mann abdominal retractor. Two patients who received spinal anaesthetics developed unilateral and one who had a general anaesthetic developed a bilateral paralysis. No further cases have occurred since 1955 when the danger from the pressure of the lateral blades in the conventional position was recognized, and the retractor was placed in a reversed position.

Acknowledgments

Appreciation is due to Doctors M. S. Ellenzweig, L. E. Lotimer, and H. R. Morgan for permission to report clinical material, and to Miss J. Ashley, photographer and illustrator of the Hamilton General Hospital, for the illustrations.

Résumé

Les auteurs présentent l'histoire des cas de trois malades qui ont présenté, au cours des années 1954 et 1955, des séquelles transitoires à la suite de blessure du nerf fémoral. La première malade a subi une hystérectomie. Elle a présenté de l'engourdissement et de la faiblesse du quadriceps gauche, signature d'une lésion du nerf fémoral, à la suite d'une anesthésie rachidienne et de l'emploi d'un écarteur abdominal Mann dans une incision transversale basse où les muscles droits n'avaient pas été divisés.

La deuxième malade a présenté un tableau semblable à celui de la première malade à la suite d'une laparatomie basse au cours de laquelle on a pratiqué une résection du présacré et on a cautérisé des plaques d'endométriose. On avait pratiqué une rachianesthésie et on s'était servi d'un écarteur de Mann de façon conventionnelle pour une incision paramédiane. La troisième malade, à la suite d'une hystérectomie sous anesthésie générale, a présenté une paralysie bilatérale du nerf fémoral. L'incision était du type trans-transverse basse.

Au début, chacun croyait que ces malheureuses présentaient des complications post-rachidiennes, même celle qui avait reçu une anesthésie générale. On a noté, toutefois, qu'en mettant en place l'écarteur de Mann dans la position habituelle pour les laparatomies, il y avait un risque que les ailes latérales fixes pointues ne compriment le muscle psoas dans la région du nerf fémoral et des vaisseaux iliaques. Le fait qu'un gynécologue ait abandonné l'usage de cet écarteur depuis qu'une de ses patientes a subi des blessures devient éloquent. Bien plus, dans les

hôpitaux de Hamilton, depuis que le docteur R. T. Weaver a suggéré d'employer l'écarteur de Mann de façon à ce que les ailes latérales portent à un endroit plus élevé et ne compriment pas les muscles psoas dans une région dangereuse pour le nerf fémoral, l'incidence de cette complication est complètement disparue.

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VOMITING, REGURGITATION, AND ASPIRATION IN ANAESTHESIA, II

BRIAN M. MARSHALL, M.D., and
R. A. GORDON, B.SC., M.D., F.R.C.P.(C), F.F.A.R.C.S., D.A.[1]

IN A PREVIOUS COMMUNICATION (1) we discussed the incidence of and the clinical and pathological findings associated with aspiration of stomach contents. We shall now consider the treatment of this complication of anaesthesia. Earlier papers on the subject of aspiration which were referred to in our previous communication stressed prevention and made little mention of specific measures of treatment.

Morton and Wylie (2) suggested ways of preventing regurgitation and suggested procedures to be used if regurgitation or vomiting occurred. These authors believed that a relaxant should not be given since it would predispose to more regurgitation. They considered immediate bronchoscopy to be "lethal."

Merrill and Hingson (3) in reviewing mortality and morbidity from aspiration in maternal cases found that prompt bronchoscopy gave excellent results in decreasing the degree and duration of morbidity.

Housmann and Lunt (4) reported three cases of aspiration in obstetric patients and drew attention to the fact that pulmonary oedema rather than bronchospasm is present. They considered post-partum adrenal insufficiency to be the major factor in the pathogenesis of the condition. This they believed to be the result of loss of the placenta secretion of adrenocorticotrophic hormone and glucocorticoids. This leads to the theory that the body cannot respond adequately to stress in the immediate postpartum period, and cortisone is therefore recommended as essential therapy in the treatment of the aspiration syndrome.

The treatment presently used in the teaching hospitals of the University of Toronto is devised essentially to establish and clear an airway and as far as possible to prevent pulmonary complications by removal of the aspirated material and prevention of pulmonary reaction by steroid therapy. The pharynx, larynx, trachea and bronchi are cleared of all regurgitated material. This must be done quickly and often in stages, as the patient must be oxygenated at the same time. To facilitate this we have found it advisable to use a fast-acting relaxant. This not only gives the anaesthetist easy access to the larynx and trachea via endotracheal tube or bronchoscope, but also prevents the patient from aspirating material into the lung periphery with gasping respirations. Bronchoscopic examination of the larynx, trachea, and bronchial tree is usually carried out. It has been found beneficial to wash out any remaining fluid and food particles with saline. If applicable, general anaesthesia is discontinued.

The chief aim of therapy is to prevent pulmonary complications following aspiration of stomach contents. Pulmonary and bronchial oedema, consolidation and atelectasis may appear within a few minutes of the accident. Pneumonitis, lung abscess, or bronchopneumonia may follow in a few hours or days. Pulmonary fibrosis, localized or generalized, may follow in a few weeks. The serious nature of this complication is exemplified in case 7, reported below. Intravenous cortisone is given immediately and continued along with ACTH for a minimum

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of three days. Antibiotic therapy, antispasmodics, and oxygen with or without aerosol inhalation are used as deemed necessary. This method has proved effective in decreasing morbidity and preventing sequelae.

The case histories presented below were collected from several Toronto hospitals. The anaesthetists were able to recall vividly much information to supplement the details on the anaesthetic charts. This was an indication to us of the importance with which the problem was viewed by our colleagues.

Case Histories

Case 1

A middle-aged female had a general anaesthetic for dislocation of the right elbow. The injury occurred at 4.00 P.M., the patient ate a meal at 5. P.M. and the anaesthetic, thiopentone sodium, N_2O and O_2, was commenced at 10.15 P.M.. At 10.30 P.M. she regurgitated and aspirated while under general anaesthesia. The patient was intubated and clear fluid and mucus were aspirated from the trachea. At 10.50 P.M. the patient was bronchoscoped. No erythema or bile staining of the tracheal mucosa was noted. The airway was clear. Scattered rhonchi were noted in both upper lung fields. The patient was admitted to hospital and given therapy of nasal oxygen and aerosol inhalations. She was asymptomatic during the night. Physical examination twelve and thirty-six hours after the accident revealed no positive chest findings. The patient was discharged. A chest X-ray taken the day of discharge, but unfortunately not reported until later, noted parenchymal infiltration of the upper lobes of both lungs (Fig. 1). This is of interest in view of the negative findings on physical examination.

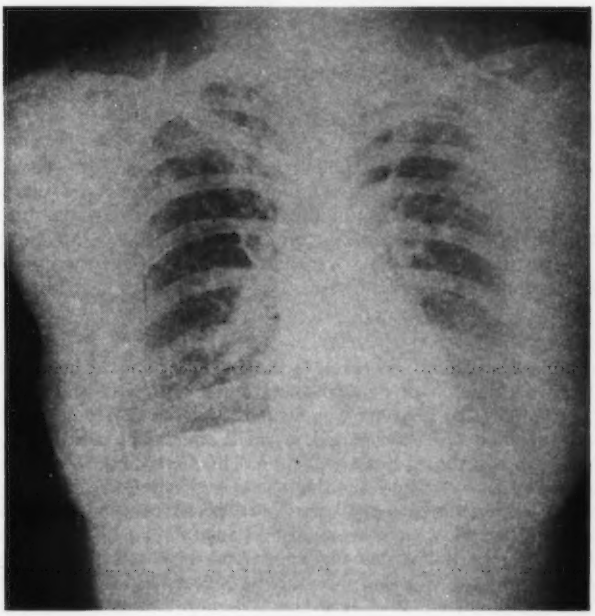
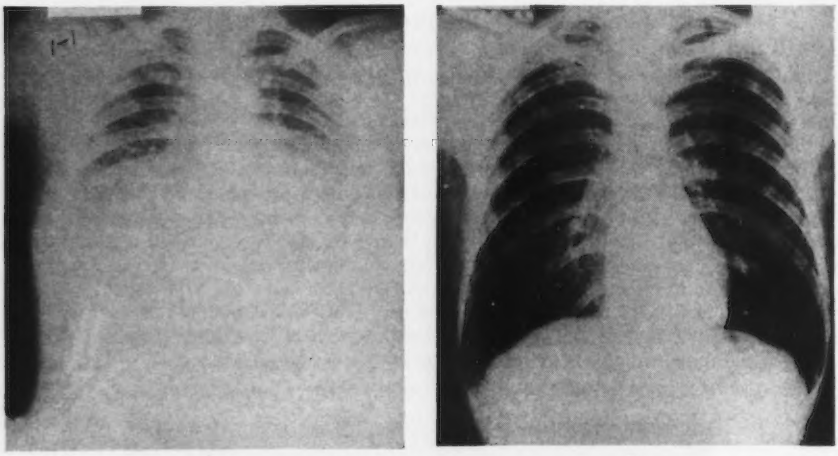


FIGURE 1

Case 2

A 40-year-old male was admitted in November, 1956, for drainage of an appendiceal abscess. Two weeks later a laparotomy was performed for small bowel obstruction. Two hours postoperatively he vomited and aspirated. The patient was immediately bronchoscoped and was given 100 mg. of Solucortef® intravenously and antibiotic therapy was commenced. A portable chest X-ray taken the following day showed "an area of infiltration in the right lower lobe in keeping with pneumonitis" (Fig. 2A). The patient was pyrexic for four days. A chest X-ray taken the day of discharge, sixteen days later, was reported to be clear (Fig. 2B).



A B

FIGURE 2

Case 3

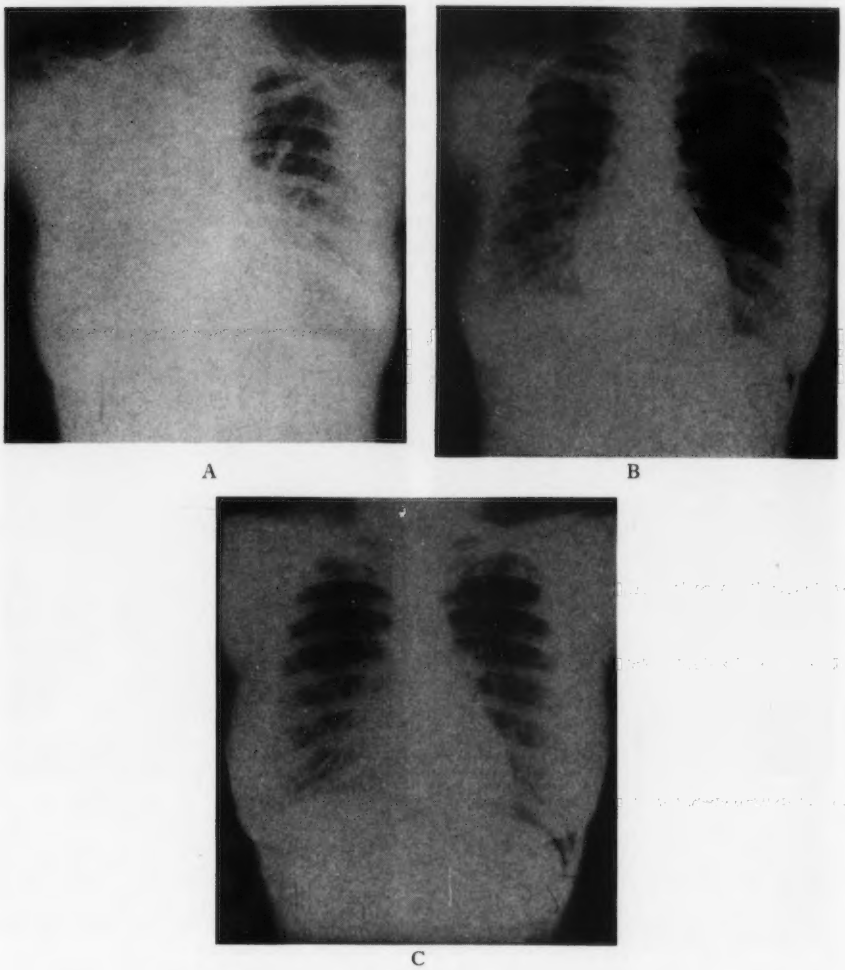
A 25-year-old primiparous patient vomited during induction of general anaesthesia for delivery. The anaesthesia was discontinued, and the pharynx was cleared. The patient delivered spontaneously. Antibiotic therapy only was given during the postpartum period. Figures 3A, 3B, and 3C are X-rays taken one day, seven days, and seven weeks postpartum. These films show the slow resolution of the infiltrative process, which was still incomplete at seven weeks.

Case 4

A young primiparous patient was taken to the delivery room 22 hours after admission to hospital. During induction of general anaesthesia with N_2O and C_3H_6 the patient vomited and aspirated. The pharynx was cleared and the patient was intubated with the aid of succinylcholine and oxygenated. A small amount of dark fluid was aspirated from the trachea through the endotracheal tube. The anaesthetic was discontinued and the baby was delivered. At this time the chest was reported clinically to be clear. Immediately postpartum the patient was given a broadspectrum antibiotic.

Twelve hours postpartum there were râles and coarse rhonchi in both lungs. A chest X-ray (Fig. 4A) was reported as showing right upper lobe and left lower lobe bronchopneumonia. At this time she was given therapy of "Compound F" 50 mg. stat and t.i.d., cortisone 25 mg. stat and q.i.d., and Aminophylline Suppositories q.i.d. This medication was continued for three days.

Twenty-four hours postpartum the patient had no clinical signs in her chest. X-rays taken seven days postpartum reported the chest to be clear (Fig. 4B).



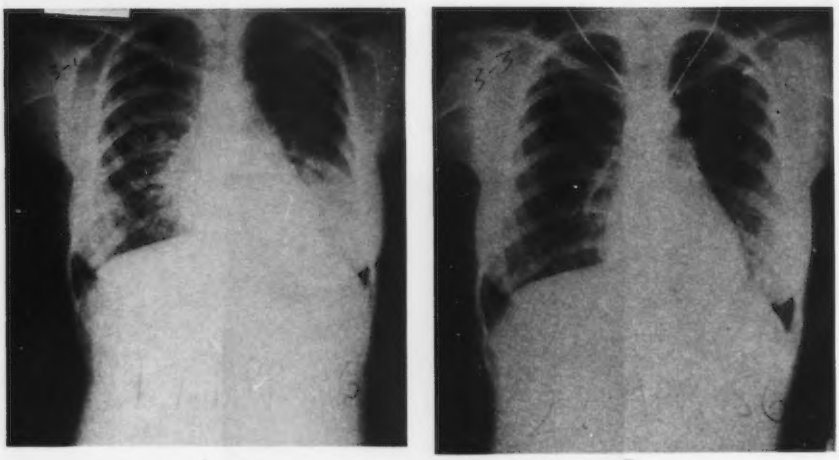
A B C

FIGURE 3

Case 5

A 21-year-old primiparous patient was presented for Caesarean section after forty-two hours of labour. Thirty minutes preoperatively the patient vomited a small amount of brownish liquid. The stomach was not emptied. General anaesthesia was induced with thiopentone. A cuffed endotracheal tube was introduced with the aid of succinylcholine. At this time a small amount of brownish fluid was noted in the pharynx, but none could be aspirated from the bronchial tree. Anaesthesia was maintained with N_2O and trichlorethylene and oxygen. The conduct of the anaesthesia was uneventful.

A few minutes after extubation the patient was noted to be slightly cyanotic. This was readily reversed with oxygen. A coarse rhonchus was present in the right chest. On the ward her condition deteriorated. Thirty minutes postoperatively râles were present in the right chest. Oxygen inhalations failed to eliminate the cyanosis. Pulse rate was 200 per minute. Moist bubbling râles were noted over the entire chest. A portable chest X-ray (Fig. 5A) showed generalized "snow storm" appearance. Hydrocortisone 100 mg. was given immediately intravenously and a further 100 mg. was given slowly during the next two hours. The patient began to improve after one hour of therapy. Her respiratory rate fell from 30 to 24 and her pulse rate from 200 to 120. The cyanosis disappeared. Râles were heard only in the right upper chest. A further 100 mg. of hydrocortisone was given during the next six hours. Metacortin was given for three days to a total dosage of 105 mg. A broad spectrum antibiotic was given for 48 hours. The patient steadily improved and was discharged on the tenth day postpartum. A chest X-ray taken on that day was reported to be normal (Fig. 5B).

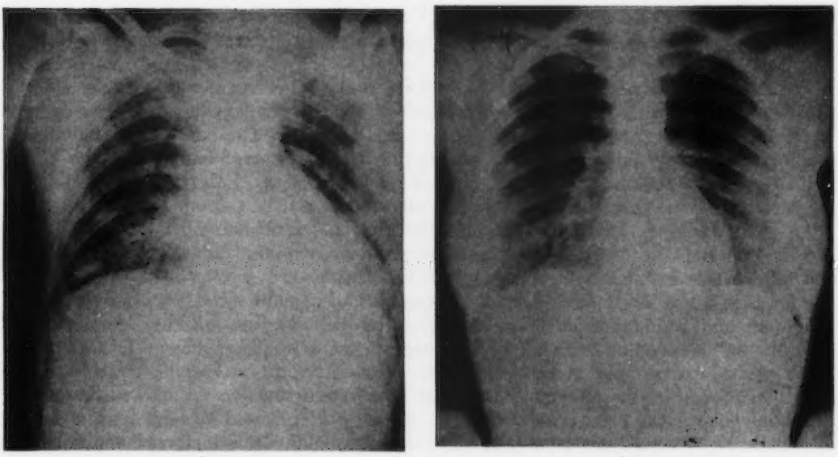


A B

FIGURE 4

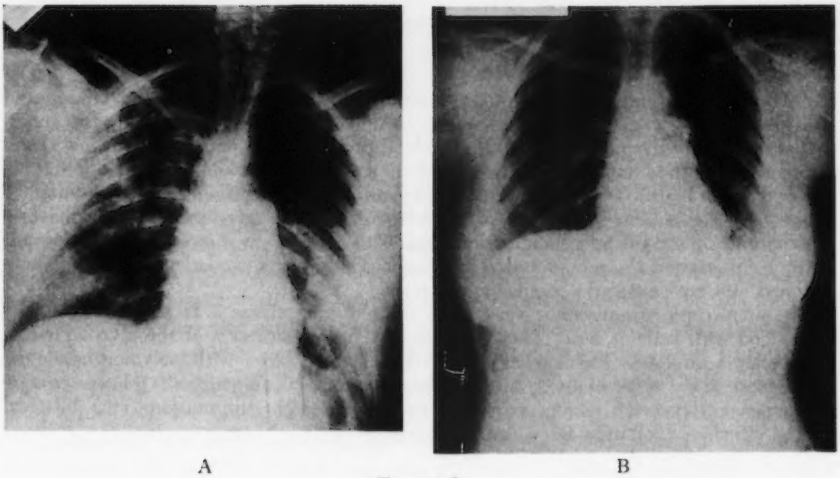
Case 6

A 21-year-old primiparous patient, in labour for sixteen hours, was delivered under general anaesthesia with N_2O, trichlorethylene, oxygen, and curare. During anaesthesia the patient vomited brownish fluid. Aspiration was suspected and she was intubated. Brownish fluid was aspirated from the trachea and bronchi through the endotracheal tube. The upper respiratory tract was lavaged with 200 cc. of saline. On physical examination the patient was dyspnoeic and had râles in both lungs. The dyspnoea and râles continued for about 48 hours. A chest X-ray taken the evening of delivery showed mottled densities in both lung fields, mainly in the lower segments (Fig. 6A). Solucortef 100 mg. was given immediately and repeated in 8 hours. One dose of aminophylline was given. Antibiotic therapy was given for three days and metacortin 10 mg. q.8.h. for one day. Although she was in an oxygen tent, the patient was dyspnoeic during the first day except when she was maintained in Fowler's position. There was steady clinical improvement, and the patient was discharged seven days postpartum. A chest X-ray taken on the day of discharge showed very little residual infiltration (Fig. 6B).



A B

FIGURE 5



A B

FIGURE 6

Case 7

A young primiparous patient had an episode of cyanosis and tachycardia during general anaesthesia for delivery. Immediately postpartum the patient was cyanotic and on physical examination was found to have a blowing apical systolic murmur, gallop rhythm, bilateral basal râles, and pitting oedema of both ankles. Her heart rate was 160 per minute and blood pressure 100/80. The heart was found to be enlarged. The patient was considered to have rheumatic heart disease with failure and was treated with quinidine, digoxin, and aminophylline. Her breathing remained laboured and difficult. Nine days postpartum she was given Cortone 25 mg. b.i.d. for six doses. A

chest X-ray taken at the end of this therapy (12 days postpartum) showed pulmonary congestion and cardiac enlargement. The patient at this time was transferred to another hospital.

Physical examination at the time of transfer showed her temperature to be 100 F., pulse rate 120 per minute, and blood pressure 90/60. She had gallop rhythm, a systolic heart murmur, râles in the left chest, and an enlarged liver. The patient responded well to digitalis therapy until four weeks postpartum when she complained of a dry cough and shortness of breath. She had one episode of haemoptysis. Following this a physical examination of the chest revealed bilateral consolidation. The patient expired one week later (five weeks postpartum). During the final stages of this illness, an ECG was reported normal, and blood and sputum cultures were non-contributory. The chest X-ray showed increased mottling throughout both lung fields.

At autopsy the lungs were heavy, oedematous, firm, and airless, with patchy consolidation and haemorrhage. There was a slight pleural effusion. The heart weighed 400 gm., and showed no evidence of valvular disease. The spleen showed infarction and there was a small infarct in the lower lobe of the left lung. No evidence of amniotic débris was found in the pulmonary arterioles. The lung tissue showed marked desquamation of alveolar living cells, patchy deposits of haemosiderin, and alveolar haemorrhages. The pathologist ended his report on the case as follows, "The cause of death in this case is obscure. It may have been a result of amniotic fluid embolism at delivery, or possibly an aspiration of gastric contents during anaesthesia." The anatomical diagnosis was: (i) diffuse haemorrhages of lungs with fibrosis; (ii) infarct of lung; (iii) cloudy swelling of myocardium.

This case almost certainly is one of regurgitation and aspiration.

Case 8

A 19-year-old primiparous patient was admitted at 8 P.M. after five hours' labour, and delivered at 3.45 A.M. the following morning. At 8.00 A.M. she was seen by the anaesthetist because of dyspnoea and a drop in blood pressure. The patient was cyanotic and dyspnoeic. She had tachycardia, hypotension, and bilateral pulmonary consolidation. She was given hydrocortisone, antibiotics, digoxin, and aminophylline, but continued to deteriorate. The patient died two days following admission, thirty-six hours after delivery.

At autopsy the right lung weighed 840 gm. The cut surface showed areas of necrosis up to 1 cm. around the moderate-sized bronchi. These areas exuded pus. The left lung weighed 490 gm. and had a similar appearance.

On microscopic examination very little aerated lung tissue was seen. The alveoli were filled with oedema fluid. There was necrosis and destruction of the alveolar walls. The oedema fluid was haemorrhagic and diffusely infiltrated with polymorphonuclear leukocytes. There were many small abscesses. Anatomical diagnosis: (i) haemorrhagic pulmonary oedema; (ii) acute aspiration pneumonia with suppuration; (iii) bilateral pleural effusion; (iv) right-sided cardiac failure.

Case 9

A primiparous patient was admitted in labour at 11 P.M. and was taken to the delivery room two hours later. The patient vomited solid food on induction of general anaesthesia. There was complete obstruction of the airway. The pharynx was cleared manually and then aspirated. The larynx was aspirated under direct vision and an endotracheal tube passed, through which the patient was alternately and gently oxygenated. Twenty minutes later the patient was bronchoscoped without the need of relaxant or anaesthetic. The bronchial mucosa was quite red, and bled when touched by instruments.

Forty-five minutes after the accident the patient was conscious. An area of atelectasis in the right lower lobe was cleared by voluntary coughing. Solucortef® 100 mg. was given intravenously over a period of 6 hours and repeated q. 12 h. ACTH 20 units was given immediately and b.i.d., and Dicrysticin 2 cc. was given immediately.

The following morning the patient was afebrile, but had an elevated pulse rate, elevated respiratory rate, and an area of atelectasis in the right lower lobe. This quickly cleared when the patient was given oxygen and aerosol (Alevaire®) inhalations. The pulse and respiratory rates returned to normal. Cortisone was discontinued the third day, and ACTH on the fifth day. The patient was discharged on the tenth day and remained well.

Case 10

A 4-year-old child came to operation with a stomach full of fruit juice and ginger ale. Actually the stomach was distended although this was not noted until the patient regurgitated and aspirated following induction of anaesthesia. Fluid was aspirated from the pharynx and the patient was intubated. Endobronchial aspiration was carried out via the endotracheal tube. The child was allowed to wake up enough to cough vigorously, but the anaesthetic was continued and the operation completed.

Four hours later the patient was bronchoscoped. There was much secretion in the bronchi. The vocal cords were red, but there were no haemorrhagic areas and the bronchial mucosa did not bleed when touched with the instrument.

At this time the child was given 100 mg. of Solucortef, followed by 50 mg. in the next 8 hours. Metacortin 30 mg. (total dose) was given over the next three days. Chloromycetin was given for three days.

At the time of bronchoscopy there were rhonchi and scattered râles in both lungs. The following day the chest was clear on auscultation, but a small area of collapse was noted in the posterior segment of the lower lobe of the right lung. The third day there was an occasional rhonchus in the right lung. On the fifth day there were no abnormal chest findings.

Discussion

The ten cases of aspiration of vomitus here described illustrate the value of the specific therapy recommended. The serious sequelae if the condition is not recognized and adequately treated may also be noted. The contrast between the treatment required for and the sequelae of aspiration of solid and liquid gastric contents is evident in comparison of Cases 6 and 9. Cases 7 and 8 show many important details. Aspiration was not recognized in either case. The rapid, fatal course in Case 8, in spite of vigorous therapy is remarkable. We feel this may be due to the fact that the aspiration was not recognized, and the anaesthetic was allowed to continue without removal of the aspirated material (cf. Case 4).

Cases 1 to 6 illustrate the difference in the pulmonary sequelae between those cases which receive inadequate or delayed treatment and those which received adequate and immediate treatment. In Case 1, although the condition was recognized, it was not considered that there was sufficient pulmonary pathology present at the time of bronchoscopy to require more specific therapy. More serious complications may have been prevented by the fact that the aspirated gastric secretions were partially neutralized by ingested food. This difference was discussed in our previous communication (1).

Conclusions

1. Aspiration of gastric contents is a serious complication of general anaesthesia and a major cause of morbidity and mortality.

2. Immediate recognition and immediate treatment are necessary to prevent prolonged morbidity or mortality.

3. Successful therapy must include prompt complete removal of any irritant from the respiratory tract and prevention of inflammatory reaction by specific drug therapy.

Outline of Recommended Treatment

Immediate

1. Stop the general anaesthetic immediately and continually oxygenate the patient while carrying out the following steps.
2. Clear the pharynx and larynx with suction and manually if necessary.
3. Prevent further aspiration by lowering the head of the table and giving a fast, short-acting relaxing agent.
4. Clear the larynx and bronchial tree by direct laryngoscopy and bronchoscopy.
5. Lavage the bronchial tree with soda bicarbonate solution or saline.

Specific Drug Therapy

1. Cortisone 100 mg. should be given intravenously immediately and 200 mg. during the next 24 hours, followed by 50 mg. twice a day for two days, or until the chest is clear.
2. ACTH 20 units twice a day for three days.
3. Antibiotics: a broad spectrum antibiotic or combination of antibiotics should be given as a preventative measure.
4. Oxygen by mask, tent, or nasal catheter should be given, sufficient to maintain adequate oxygenation.
5. Aminophylline intravenously or in suppositories may be given.
6. Expectorant cough mixtures may aid in the production of thin easily removed bronchial secretions.
7. Inhalation therapy with a detergent aerosol may be beneficial in the prevention or treatment of atelectasis.

Acknowledgments

We wish to thank our colleagues of the Departments of Anaesthesia of the other hospitals within the University of Toronto Teaching Group who have co-operated with us in the preparation of this study by making available to us the details of several of the case histories reported.

Résumé

Au cours d'un exposé antérieur, les auteurs ont parlé de la fréquence et des séquelles cliniques pathologiques de l'aspiration de liquide gastrique. Dans cette communication-ci, il sera question du traitement de cette complication de l'anesthésie avec l'anamnèse des histoires de cas de dix malades.

L'aspiration de liquide gastrique est une complication sérieuse de l'anesthésie générale et une cause fréquente de morbidité et de mortalité. Pour prévenir la morbidité et la mortalité, il s'impose de reconnaître le fait immédiatement et de pratiquer le traitement aussitôt. Dans un traitement efficace, il faut inclure

l'ablation précoce et complète de toute substance irritante dans les voies respiratoires et faire la prévention d'une réaction inflammatoire par les médicaments appropriés.

Traitement suggéré

Immédiatement

1. Suspendre immédiatement l'anesthésie générale et continuer à oxygéner le malade tout en faisant ce qui suit.
2. Nettoyer le pharynx et le larynx avec un aspirateur et avec les mains si nécessaire.
3. Prévenir une nouvelle aspiration en abaissant la tête du malade et en administrant un myorésolutif à action rapide et courte.
4. Nettoyer le larynx et l'arbe bronchique en faisant une laryngoscopie directe et une bronchoscopie.
5. Faire un lavage de l'arbe bronchique avec une solution physiologique ou bicarbonatée.

Médication spécifique

1. Administrer immédiatement par voie endoveineuse de la cortisone 100 mg. et, durant les 24 heures suivantes, 200 mg.; ensuite 50 mg. b.i.d. durant deux jours ou jusqu'à ce que le poumon soit clair.
2. ACTH 20 unités b.i.d. durant trois jours.
3. Des antibiotiques. Comme mesure préventive, administrer un antibiotique à grand spectre ou encore une association d'antibiotiques.
4. Donner, soit par masque, tente ou cathéters nasaux, de l'oxygène en quantité suffisante pour maintenir une bonne oxygénation.
5. On peut aussi donner de l'aminophylline par voie endoveineuse ou en suppositoires.
6. Des mixtures expectorantes peuvent aider à éliminer des sécrétions fluides et faciles à expectorer.
7. On peut aussi, en prévention ou comme traitement d'atélectasie, faire inhaler un détergent par aérosol.

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NEWS LETTER

Annual Meeting*

The 1958 Annual Meeting at the Seigniory Club was a resounding success in every respect. Registration was the largest in history: 197 anaesthetists and 73 ladies. The clinical and scientific presentations of the programme were of the highest merit, and it was evident that much more time might have been profitably used both in discussion and in presentation. Council recognized this fact in extending the 1959 meeting to four days.

The business of the Society required meetings of Council on Sunday, June 22,

Seen at Dance

*Pictures by courtesy of Mr. Gordon Staples.

Seen at Dance

Monday, June 23, and Wednesday, June 25, while the Annual General Meeting required two sessions for completion of business.

Despite all this serious side of things, however, there was time for play and relaxation. The fairways and greens of the Seigniory Club course were well populated by golfers, early and late. The ladies' tea on the terrace attracted many of the gentlemen, while others were busy acquiring saddle sores (or bruises) or demonstrating the latest version of the dog-paddle in the swimming pool.

The traditional cocktail party, dinner, and dance was "sold out," and the affairs of the evening covered the whole spectrum of formality and informality from black ties and the installation of the President to beach suit attire in honour of the British Columbia Centennial and the vision of the Assistant Secretary cooling her aching feet in the fountain.

This meeting was an important and pleasant occasion: an occasion to share the best in Canadian Anaesthesia: an occasion to meet old friends and to make new ones. Those who had this experience will be looking forward to renewing it in the same location from May 4–7, 1959.

Dr. Eugene Allard addresses the Annual Dinner after his installation as President.

Saskatchewan Division

Dr. A. R. Deacock, late of King's College Hospital, London, England, and Dr. J. H. Harland of Vancouver have joined the Department of Anaesthesia of the University of Saskatchewan as Instructors.

Dr. E. T. Thomas has left the Department of Anaesthesia of the University of Saskatchewan to return to England.

Dr. A. K. Bradshaw of Edmonton, Alberta, has been appointed Research Fellow in the Departments of Anaesthesia and Medicine at the University of Saskatchewan.

Dr. K. E. Lee has passed the examination for the F.A.C.A. after 18 months of residency training at the University of Saskatchewan.

Dr. Otto Smith has completed his second year of residency training at the University of Saskatchewan and is now practising with Dr. J. Wishart and Dr. D. Aiken in Peterborough, Ontario.

MEETINGS

CANADIAN ANAESTHETISTS' SOCIETY
Annual Meeting, Seignory Club, Montebello, P.Q.
May 4–7, 1959

Western Divisions

CANADIAN ANAESTHETISTS' SOCIETY
Bessborough Hotel, Saskatoon, Sask.
March 19–20–21, 1959

INTERNATIONAL ANESTHESIA RESEARCH SOCIETY
33rd Congress, Deauville Hotel, Miami Beach, Fla.
April 20–23, 1959

WORLD CONGRESS OF ANAESTHESIOLOGISTS
Royal York Hotel, Toronto, Canada
September 4–10, 1960

BOOK REVIEWS

PHYSICS FOR THE ANAESTHETIST. By Sir Robert Macintosh, William W. Mushin, and H. G. Epstein. Second edition. Springfield, Ill.: Charles C. Thomas [Toronto: The Ryerson Press]. 1958. Pp. 443. $18.50.

The first edition of this work, published in 1947, filled a great need in the anaesthetic literature, and after being out of print for five years has become almost a collector's item. This second edition has been eagerly awaited by practising anaesthetists, teachers, and students alike.

This textbook has grown from 235 pages in the first edition to 443 in the new edition. The major part of this expansion results from the addition of a very welcome chapter on "Pressure Reducing Valves," and four excellent chapters on "Explosions." The authors make an admirable job of the task of applying the principles of elementary physics to the problems of anaesthesia. The book is lavishly illustrated, and undoubtedly may be fully understood even by those who have long ago forgotten what they learned of Physical Science during earlier training.

R.A.G.

BLOOD VOLUME DETERMINATIONS WITH RADIOACTIVE ISOTOPES AND INDEX OF CARDIAC CLEARANCE. Washington: United States Atomic Energy Commission AECU-3614. March 1958. $1.00.

The use of radioactive isotopes as a tool in the investigation of problems of the circulation is a matter of interest to many medical research workers. This publication is based on investigation in the Department of Anesthesiology Research Laboratory at the District of Columbia General Hospital, Washington, D.C., and discusses the principles and techniques involved in the employment of isotopes in measurements of blood volume and index of cardiac clearance. It may be obtained from the Office of Technical Services, Department of Commerce, Washington, D.C., U.S.A.

R.A.G.

CANADIAN ANAESTHETISTS' SOCIETY JOURNAL

Volume 5

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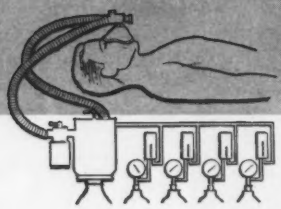

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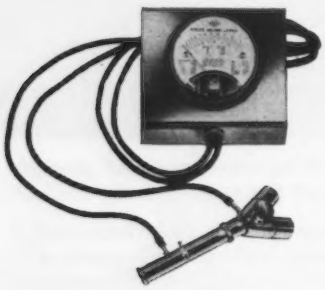

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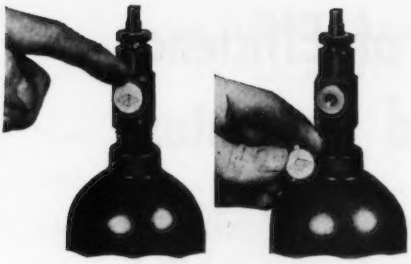

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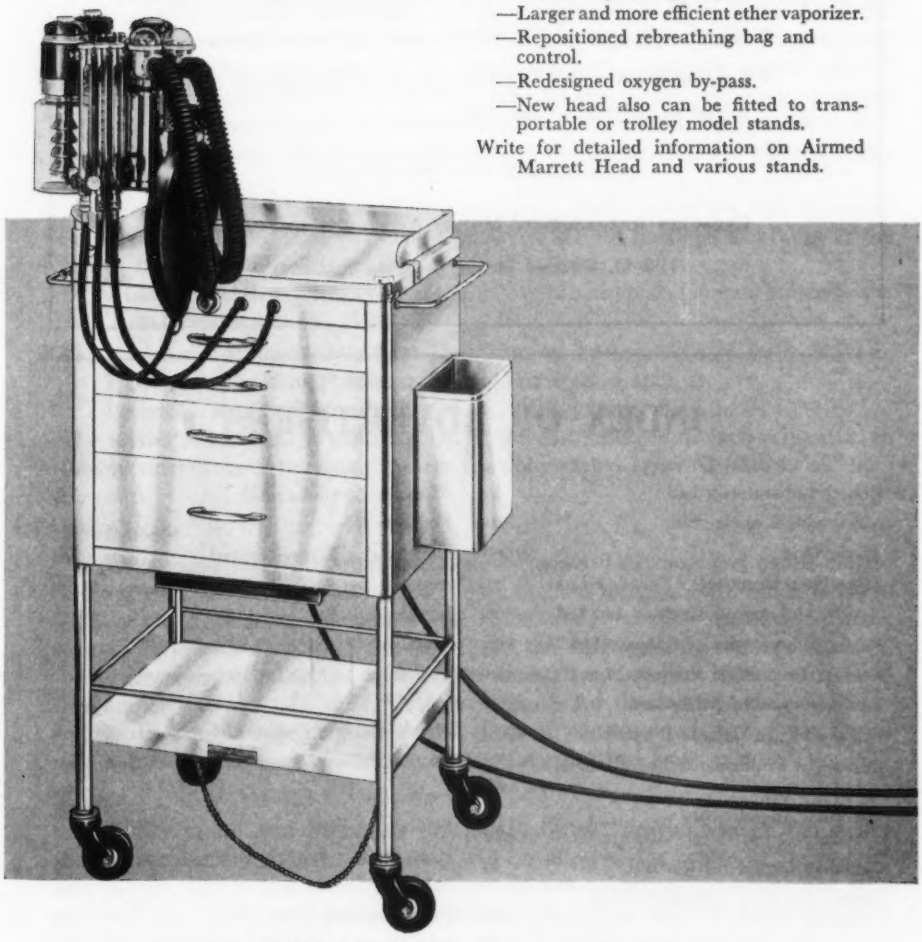

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